BRAIN.

PART IV., 1906.

Original Articles and Clinical Cases.

CASE OF TUMOUR OF THE MIDDLE LOBE OF THE CEREBELLUM — CEREBELLAR PARALYSIS WITH RIGIDITY (CEREBELLAR ATTITUDE) — OCCASIONAL TETANUS-LIKE SEIZURES (1871).

BY J. HUGHLINGS JACKSON, M.D., F.R.S.

In this article is reproduced a paper which was published in the *Hospital Reports* of the *British Medical Journal*, November 4, 1871. Unfortunately, a sketch of this patient's attitude in the state of paralysis with rigidity and another sketch of him whilst he was having tetanus-like seizures, sketches exhibited when the paper was read, were not published with the report (*op. cit.*). The sketches were made by Sir Stephen Mackenzie (called Mr. Stephen Mackenzie in the following report), and are now given. To Sir Stephen I am greatly indebted also for valuable clinical observations on the case.

The reader is asked to bear in mind that this patient's case was investigated by Sir Stephen Mackenzie and myself many, thirty-five, years ago. The subject was then new to us. My own remarks (1871) preceding the narrative of the case, and those concluding the paper (1871) are old-fashioned in the wording. (Words within square brackets are added in 1906).

"I wish in this communication [1871] simply to record a case of clinical interest, and not to make the case a text

for remarks on the functions of the cerebellum. This is the fifth case of disease of the cerebellum which I have had under my observation since January. The symptoms in these five cases were so different, that I am less inclined than ever I was to make general remarks on cerebellar disease. As in lesions elsewhere, we have to classify our cases of cerebellar disease with especial regard to the suddenness or slowness of the lesion. Cases of cerebellar hæmorrhage and cases of tumour of the cerebellum *seem* to differ even in kind. The case which I relate is one of tumour, and of tumour so placed that the diagnosis was comparatively easy.

"John R., aged 5, was admitted April 25 [1871], and was afterwards transferred to my care by Dr. [later, Sir] Andrew Clark. Copious and very careful notes of the case were taken, chiefly by Mr. [now Sir] Stephen Mackenzie; and of these the following is an abstract.

"*History.*—The boy had cut his teeth early. He walked at the age of thirteen months, and, except for two convulsive seizures, both on one day, when (his mother said) he was cutting a top tooth, he had been healthy. Four months before admission, the illness of which he died began. The first things noticed were, that the boy's head became so much larger that his father's cap would fit him, and that he complained of pain at the back of his head. He cried a good deal, and lost flesh. Two months later, he began to be 'stupid.' Fourteen days before admission, his legs began to fail in walking [*see* later a note on reeling, under the heading 'State of Limbs']; and, two or three days before admission, he began to pass urine and fæces without notice."

When admitted, he seemed to be in good general health. He took his food well; and, although his mother said he had become thinner, he was well nourished. In what follows, the particulars of his case will be arranged under the headings: (1) Mental Condition and Size of Head; (2) State of Optic Nerves and Condition of Sight; (3) State of Limbs; (4) Convulsive [tetanus-like] seizures.

"(1) *Mental Condition and Size of Head.*—His mental

condition was very peculiar. He very rarely spoke, except when spoken to, or when he wanted something to eat or drink; but he did talk well on these occasions. He had picked up a phrase since his admission into the hospital. When any one asked him 'What does the dustman say?' he would give a remarkably exact imitation of the dustman's cry, 'Dust a-hoy.' At a later date he sang, the nurse told us, in good time, two lines of a hymn; and, when Mr. Stephen Mackenzie asked him to sing it again, he got out the words 'Angels of Jesus,' pitching his voice properly. His memory was very deficient; he could not remember the name of his next bedfellow; did not care to see his father or mother; and it was doubtful if he recognised his mother. He did not play. He was, however, very good-tempered, and, as the nurse said, 'biddable.' Indeed, for his age, he was abnormally good-tempered—to a degree of apathy. His general mental condition is best described by the word Mr. Mackenzie uses in his report—'hebetude.' There was a general [and presumably a very slow and gradual] fading out of mental power. [See remarks on 'fading out of mind' and on hydrocephalus in the next article]."

He had a large head, but there was nothing characteristic about its enlargement. The orbital plates were not depressed; no gaping of the sutures could be detected; and there was no great prominence above the ears. It did not become much larger before death. There was no evidence of rickets in his history, nor were there any relics of it in the shape of his chest.[1]

Optic nerves and sight.—At first his sight was not obviously affected, but it was impracticable to test it [par-

[1] [1906. The following is from a paper I published, *British Medical Journal*, February 7, 1880]:—

"There are indirect symptoms from tumour of the middle lobe [of the cerebellum] and large tumours under the tentorium. There is vast increase of fluid in the lateral ventricles of the cerebrum in some cases. In children, the head enlarges; there is great hebetude of mind, a slow failure of mental faculties. This [increase of fluid] I used to suppose to be caused by pressure on the veins of Galen. Dr. (now Sir) Stephen Mackenzie, in an admirable paper on 'Cerebellar Disease,' recently read before the Hunterian Society, suggests as an additional and more important factor closure of the communication between the ventricles and the subarachnoid space."

ticularly]. Neither the boy's mother nor his nurse supposed that there was anything the matter with it, although Mr. Mackenzie found that there was double optic neuritis. On May 11, when I for the first time used the ophthalmoscope, there was no obvious imperfection of sight. Both optic discs were much swollen and raised; the arteries were concealed in the swelling; the veins were large and partly obscured, "knuckling" over the edge of the disc. The surface of the disc was of a nacreous appearance. There were no effusions of blood, nor did any appear afterwards. He subsequently became blind. The discs gradually cleared up to a remarkable extent, and on the day before his death they were of little more than normal size, but were indistinctly edged and a little swollen. Since headache and vomiting often occur with this pathological condition of the discs, it is not out of place to mention here that, during the boy's stay in the hospital, he did not seem to have pain in the head. He vomited frequently, but had no dyspepsia, taking his food well almost to the last.

"(3) *Limbs.*—When I saw him he could not walk by himself, but, when helped a little, could reel along. His gait was a reel, not a stagger like that of ataxy; nor was it the gait of paraplegia. Whenever left to himself during the trial of his walking, he fell to the side to which he was inclining."

[1906. I believe that the reel in an early stage of disease of the cerebellum is owing to weakness of the muscles (properly loss of some of their movements), which brace up the spine, a suggestion made long ago by Niemeyer. It is true that the legs in an early stage of cerebellar disease, when there is reeling, act erratically, but they are not in fault. To speak metaphorically, they are not only doing their duty, but are doing more than what is in health required of them; the abnormalities of their motions are praiseworthy attempts to prop up the trunk and, so to say, "to run after it" in the various over-inclinings of it which are consequent on loss of some of the movements of the spinal muscles. The motions of the legs in a reeling man are better called "super-normal" than "abnormal."]

"Subsequently to the fits [tetanus-like seizures] (to be presently described) both legs became permanently stiffened in a line with his body, the feet being strongly extended, and to a trifling extent turned in. As will be seen, this permanent set of the boy's legs was like their condition in the fits [tetanus-like seizures] which preceded them.

"(4) *Convulsive seizures.*—[1906. I now call such seizures as this patient had 'tetanus-like seizures'; 'tetanoid' would imply a resemblance to either 'tetanus' or 'tetany']. The first was on May 5. The nurse called these 'shocks,' and said that both legs became rigid. He had many of this kind, but at very irregular intervals. Several of these seizures were carefully watched by Mr. Stephen Mackenzie. The following is Mr. Mackenzie's concluding report of the case, and contains an account of several seizures which he saw.

"July 11. There had been no material change in his condition for some time, except a progressive loss of flesh. He was now much emaciated. His mental condition continued of the same character. He lay still and never spoke unless he was spoken to; and his replies were always monosyllabic. He now could utter another street cry—'Any watercresses?'—in good intonation, and, when saying it, smiled, which he never did in saying anything else. When he was asked who any one near him was, he invariably replied 'Grandmother'; and, in answer to the question 'How are you to-day?' he invariably replied 'Nothink.' The great test of his bodily strength each day had been the degree of vehemence with which he could utter 'Dust-a-hoy.' To-day his voice was piteously feeble. He was much weaker, but still took his food well. He had had convulsive [tetanus-like] seizures every few days, several of which Mr. Mackenzie saw. Sometimes, but not always, the seizure was preceded by a loud cry. There was no marked twitching of the face, nor any special deviation of the eyeballs. His hands were clenched; his forearms were flexed on the upper arms, which were generally kept to the sides. The head was drawn back and the back was curved. His legs were always extended to the fullest possible degree

the feet being arched backwards. Sometimes he passed fæces and urine in an attack. The seizures generally lasted about three or four minutes, and, when passing off, they returned if he were moved about. He was not unconscious. There was no chronic spasm. In one attack the two eyes were turned to the left, and the head to the right [the head], not backwards, as usual.

[1906. The following is a sketch by Sir Stephen Mackenzie of the boy when in one of his tetanus-like seizures, and is presumably of the seizure just described. The attitude depicted in that sketch (and so for the second sketch) is a caricature, with gross exaggeration in some particulars, of the attitude depicted in a snapshot of a man or a child running very fast. In running very fast the extensor muscles of the spine are in strong action to prevent the forwardly-inclining trunk from collapsing—to save the runner from tumbling on the ground with his face downwards; the opisthonos in tetanus-like seizures is a gross exaggeration of the activity of the extensor muscles of the spine in swift running. The position of the arms in tetanus-like seizures from tumour of the middle lobe of the cerebellum is very like their position in swift running; in swift running the arms are in alternate action (note the position of the arms in the first figure). Observe that there is acute flexion at the elbows, both in severe tetanus-like seizures from cerebellar tumour (second figure) and in fast running. In this connection I beg the reader's attention to the position of the arms in a *severe* seizure of *surgical* tetanus. A drawing of a man in a *severe* seizure of surgical tetanus (the drawing is from a painting by Sir Charles Bell), is given by the late Dr. Ross in his *Diseases of the Nervous System*, second edition, vol. ii., p. 799. I quote what Ross wrote of the arms in such a seizure: "When the spasm extends to the muscles of the upper extremities, flexion predominates over extension; and during the paroxysms the arms are drawn close to the chest; the forearm is flexed upon the arm; the hand is flexed at the wrist; and the fist is closed, the palm being directed towards the upper arm." The hands are clenched in swift running; professional runners carry corks in their hands when racing.

TETANUS-LIKE SEIZURE

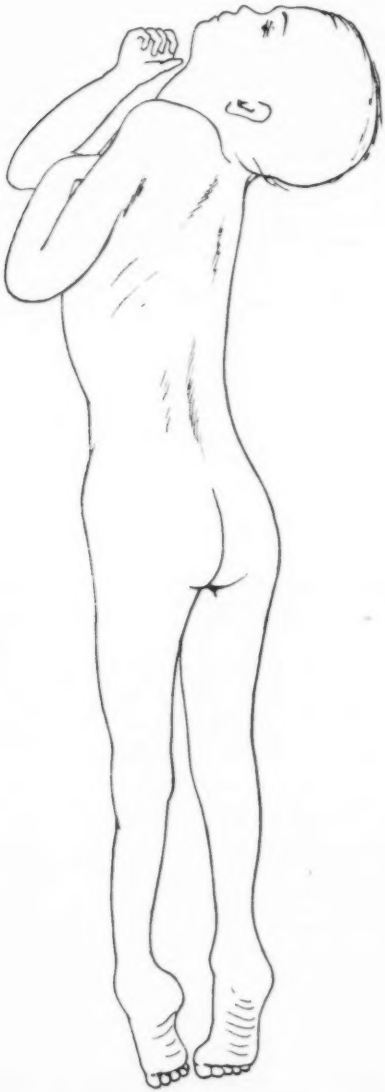
(Case of Tumour of the Middle Lobe of the Cerebellum).

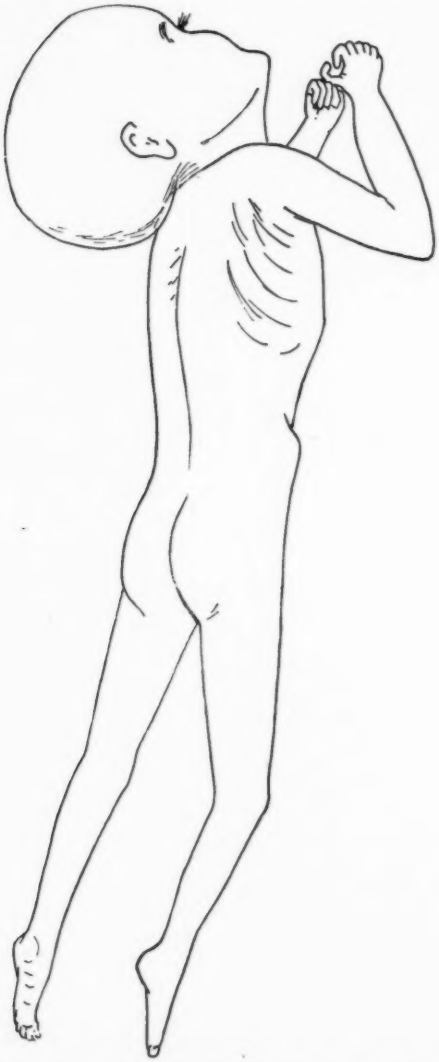
Here consider the position of the feet both in tetanus-like seizures and in swift running. In the latter there is a succession of springs from toe to toe; in the sketches in this paper there is depicted the "toe-attitude" of the feet during the tetanus-like paroxysms. I hope shortly to publish an article on the comparisons and contrasts which may be made between (1) degrees of locomotion from walking to swiftest running; (2) degrees of tetanus-like seizures in some cases of cerebellar tumour, and (3) degrees of paroxysms of ordinary surgical tetanus. The order of development of movement in 2 and 3 is but a brutal caricature of the order of development in 1. (In each the order of development of movements is a compound order.)]

[1906. Tetanus-like seizures vary to some extent on different occasions in the same patient. The following sketch, by Sir Stephen Mackenzie, is of another of the boy's seizures.]

"July 21. For the last three days the patient had been much worse. He had been lying during this time in almost exactly the same position—partly on his chest, and partly on his left side, lying askew. His back was arched backwards; the head was retracted apparently in the greatest possible degree, and the legs were fully extended. [Persisting Cerebellar Attitude.] But now and then there was a perceptible increase in the rigidity [probably, as they occurred 'now and then,' they were slight tetanus-like seizures]. He could not swallow either solids or fluids, but answered simple questions. His 'Dust a-hoy' was painfully feeble. He died the next day. [The following is a drawing by Sir Stephen Mackenzie, showing the patient's persisting attitude (cerebellar paralysis with rigidity, that is, a Cerebellar Attitude); that attitude was, in general appearance, like that in the patient's tetanus-like seizures, but the process of causation of the two conditions is, in my opinion, very different].

"*Necropsy.*—The body was much emaciated, but no disease was found in any part of the body except in the head. The head was large, the cranial bones were thinned, and the sutures were loose; the convolutions were flattened,





CEREBELLAR PARALYSIS WITH PERSISTING RIGIDITY

("Cerebellar Attitude" in a Case of Tumour of the Middle Lobe of the Cerebellum).

and the cerebral ventricles were distended by fluid; the horns of the lateral ventricles were much dilated. There was a growth like that [*vide infra*] in the cerebellum, involving, and, as it seemed, replacing the right corpus albicans. The tentorium was much bulged upwards, and under it was found a tumour of the middle lobe of the cerebellum, of about the size of a billiard ball. The corpora quadrigemina were much flattened, and the veins of Galen were greatly dilated. The tumour, on section, was of a grey-green colour. It was rounded, easily separable. Its surface was defined and vascular. Dr. Moxon was so kind as to examine the tumour for me. He concluded that it was tubercle, partly from the facts stated, partly from the microscopical structure, and partly from the age of the patient and the absence of evidence of syphilis in the clinical history. He reported on the microscopical structure as follows: "Though it presents a great variety of elements in small quantity, yet by far the main part consists of the small rounded corpuscles that usually characterise tubercle, better formed and varying corpuscles being in the outermost part, and the 'tubercle corpuscles' deeper."

"REMARKS.—[1871]. The diagnosis in this case was not difficult. There were three chief symptoms: (1) enlargement of the head; (2) blindness; and (3) reeling gait. Each of these symptoms, by itself, would be of uncertain value in diagnosis. The enlargement of the head might be owing to an [to a voluminous] adventitious product in the cerebrum—a large hydatid cyst, for instance. In such a case [in a young child] there might be an irregular gait, very like the reeling which was observed in the boy's case; but there would be, if not decided hemiplegia, much more weakness of one side of the body than of the other. The blindness from optic neuritis was of no value as to the position of the lesion, because tumour, in very many parts of the encephalon, gives rise to double optic neuritis [and thereby to defect or loss of sight]. Perhaps, of the three symptoms, the reeling gait was of most value, but by itself would only point to tumour under the tentorium [would only localise to that extent]. Putting the symptoms together it was supposed that there

was a tumour of the middle lobe of the cerebellum, which pressed on the veins of Galen and straight sinus, and thus caused, so to speak, 'ascites of the brain' (hydrocephalus). I made this diagnosis with confidence, because I had, some years ago, seen a case almost quite like that of my patient under the care of Dr. [afterwards Sir William] Gull. Dr. Gull, without any hesitation, diagnosed tumour of the middle lobe of the cerebellum, and this was found *post mortem*. Again, I made a diagnosis in a similar case a year ago—correctly as to the position of the disease, altogether incorrectly as to its nature. Lastly, a few months ago Mr. Waren Tay showed me a case in which, from the presence of like symptoms, he correctly diagnosed tumour of the middle lobe of the cerebellum."

"[1871 continued.] The symptom which is the most interesting in the case is the convulsive seizure [tetanus-like seizure]. I regret exceedingly that I have mislaid a sketch of the boy's position in one of the severest of these seizures. [It was afterwards found and is reproduced in an earlier part of this 1906 reprint from the *British Medical Journal* of 1871]. The back was much more arched than is represented in the drawing handed round. It is important to contrast this seizure with those which are more often seen from tumour of the cerebrum. In these cerebellar convulsions [tetanus-like seizures], the spasm was chiefly tonic. In cerebral convulsion it is chiefly clonic. In this [the boy's] case, the convulsion affected most the bilateral [bilaterally or alternately acting] muscles of the trunk and the legs, whilst in cerebral convulsions the unilateral [in most operations unilaterally acting] muscles are most affected, and the arms are more affected than the legs. In the kind of spasm and in its regional distribution, it [the boy's seizure] resembled rather tetanus ["surgical tetanus"] than epileptiform convulsions [Bravais, epilepsy]; and although the paroxysms differed from what is most commonly seen in the paroxysms of tetanus, it was enough like to serve as a fragment of evidence in support of the view that the [morbid] changes in tetanus are in the cerebellum. [1906 not limited to this organ.] The cerebellum seems to repre-

sent the muscles of the body in an order the reverse of that in which they are represented in the cerebrum. The current view is, that the cerebellum co-ordinates more especially those movements which serve in locomotion and other quasi-automatic processes; whereas the cerebrum co-ordinates more especially the movements which serve in voluntary operations.

[1906. In another paper will be dealt with what I suppose are motorily the anatomico-physiological relations of the cerebral and cerebellar Sub-systems. Anatomically, the representation by each Sub-system is of the whole of the skeletal musculature; the two representations are by opposite, but yet corresponding opposite, sets of movements; physiologically (dynamically), there is antagonism between them ("amicable antagonism"). In some remarks now (1906) appended to the reproduction of this (1871) paper I refer to observations by other physicians on what I have called tetanus-like seizures in cases of cerebellar tumour.]

[1906.] It will be understood that there are many cases of tumour of the middle lobe of the cerebellum without the rigidity called Cerebellar Attitude, and also without Tetanus-like Seizures.

[1906.] Dr. Dreschfeld reports (*Journ. of Anat. and Phys.*, April, 1880, vol. xiv., pp. 337 *et seq.*) a case of cerebellar tumour (superior vermiform process), interesting in many ways, and to me particularly on account of the tetanus-like seizures. Dr. Dreschfeld refers to a case recorded by Dr. Bacon, in which also there were tetanus-like seizures. The following is an extract from Dr. Dreschfeld's paper on the case of his own patient (I italicise some words): "The symptoms remained the same till the beginning of May, when the patient had an epileptic attack, which was followed by many more in the progress of the disease. These attacks, several of which were witnessed by myself and Dr. Milne, *were much more of a tetanic character than ordinary epileptic seizures.* With or without loss of consciousness, the four extremities became suddenly powerfully contracted; *the arms flexed*, the legs extended and the head forcibly drawn back. This state was followed by slight clonic con-

tractions of very short duration, and to these succeeded a state of complete exhaustion which lasted several hours." Dr. Dreschfeld writes in one part of his commentary on this case: "The tetanic nature of the convulsions, which was so well expressed, and so peculiar as to arrest our attention, derives great interest from the very recent observations of Hughlings Jackson (*British Medical Journal*, February 7, 1880), according to which rigidity of groups of muscles and tetanic-like paroxysms form important localising symptoms in cerebellar diseases." "Since the publication of Hughlings Jackson's interesting paper, a case by Dr. Bacon (*British Medical Journal*, February 21, 1880), of cerebellar tumour has been reported, in which such tetanic seizures were well marked; and I have at present a patient who, besides the ordinary symptoms of cerebellar disease, has persistent retraction of the head, owing to rigidity of the muscles of the neck. Again, stiffness of limbs has been found to follow ligature of both vertebrals in animals (Cooper, Schiff)."

[1906.] In the Dublin *Journal of Medical Science*, April, 1880, vol. lxix., p. 287, Dr. David Drummond records a case of tumour of the cerebellum. On one occasion this patient, a boy, aged 6 years, had a seizure thus described, p. 288, "during a period of severe headache, which was characterised by more intense pain and nearer approach to unconsiousness than usual; it was noticed that the spinal column was completely arched (opisthotonos). This condition of rigidity lasted for fifteen or twenty minutes, and then for three or four minutes clonic spasm of the right arm and leg ensued. At the same time the right eye was markedly turned inwards, the pupil being almost hidden."

[1906.] In reference to cerebellar attitude, the reader is referred to a case I recorded in a paper published, *British Medical Journal*, May 15, 1875, p. 636, one with the strange assortment of symptoms mentioned in the title of that paper, *On Choreal Movements and Cerebellar Rigidity in a Case of Tubercular Meningitis*. The patient was a boy, aged 4½ years, admitted into the London Hospital, February 27, 1873. It is the only case of tubercular

meningitis in which I have seen movements of chorea. I mean movements quite like those in ordinary chorea, the chorea of Sydenham. In the note I now write nothing more will be said of the chorea; indeed, little will be said of the rigidity beyond giving quotations to show what I have long ago meant by the term Cerebellar Attitude or Cerebellar Position. I find that in the quotations the term cerebellar position was used. I have pluralised some words as they ought to have been in the original, acknowledging these changes by italics.

[1906.] The first quotation from the 1875 paper is about the cerebellar attitude in general without reference to the meningitis case, but with regard to cerebellar tumour: "In some cases of disease of the middle lobe of the cerebellum, the patient lies on his back, with his legs and feet extended, the latter a little turned in; the *arms are* acutely flexed at the elbow; the *wrists are* flexed, and the *palms are* turned towards the front of the upper arm. The head may be thrown back. The spasm is tonic."

[1906.] In a continuation of the quotation from the 1875 paper, I speak of tetanus-like seizures, not referring to the case of meningitis, but to such seizures in cases of tumour of the middle lobe of the cerebellum. "In some cases of tumour of the middle lobe of the cerebellum, besides this permanent set of the limbs and body, there occur paroxysms of spasm very like those of ordinary traumatic tetanus."

[1906.] The following quotation from the 1875 paper deals once more with the Cerebellar Attitude (again called cerebellar position), but this time as manifested in the case of meningitis, under the date of March 7. Dr. Hughlings Jackson made the following note: "His two arms are now in the 'cerebellar position' [flexed at the elbow]; and so, I am told, they have been all day and night, except that once he moved the left downwards. The arms are tremulous; the tremor is occasional, and as if an attempt at slight movements. There is no frown on his face. His masseters are very rigid. His legs are kept

straight out; feet extended (he was lying on his back.) His belly is much retracted, but the abdominal wall is not stiff. He occasionally puts his left arm down slowly and vaguely, but it soon goes up again; The right arm is rigid. The elbows are kept to the sides." [1906. The retraction of the belly was, no doubt, part of the symptomatology of the meningitis; the abdominal walls were not rigid.]

CASE OF TUMOUR OF THE MIDDLE LOBE OF THE CEREBELLUM. CEREBELLAR ATTITUDE. NO TETANUS-LIKE SEIZURES. GENERAL REMARKS ON THE CEREBELLAR ATTITUDE (1872).

BY J. HUGHLINGS JACKSON, M.D., F.R.S.

[1906.] THE particulars of this case are from the Hospital Reports of the *British Medical Journal*, 1872; it was reported in two instalments, July 20 and August 3 of that year. The sketch now supplied was not published with the original (1872) report. The sketch is of value because it shows some conspicuous features of what I have called the Cerebellar Position, and, more recently, the Cerebellar Attitude, which is cerebellar paralysis with cerebral influx. It does not show all the features of that attitude. We cannot expect quite similar results in different cases from the quasi-random doings of tumours growing in the same organ. As stated in the report of the case (1872), and as will be seen by Sir Stephen Mackenzie's sketch, there was no drawing back of the patient's head and no arching of the back. The disposition of the arms depicted is, I think, what is most common in the cerebellar attitude produced by tumour of the middle lobe of the cerebellum; it differs, but not in fundamental character, from the disposition of those limbs seen in the third sketch given in my former article (case first published in 1871). I ask readers to bear in mind that the case, now republished, was investigated, and that the remarks upon it were made many, thirty-four, years ago.

[1906.] It is important to note that there were no tetanus-like seizures in the patient's symptomatology. As said in my former paper, in some cases there is neither a cerebellar attitude nor are there tetanus-like seizures. The tumour of the middle lobe of the cerebellum, by destroying, produces paralysis only, the rigidity fixing the

attitude is the result of cerebral influx, that influence being no longer antagonised, or being less antagonised, by cerebellar influence. Tetanus-like seizures are, I submit, owing to changes of instability, set up in nerve cells near the tumour—are owing to a cerebellar discharge lesion; the tetanus-like seizures are owing to occasional intense discharges of this persisting discharge lesion. Now for what was published in 1872.

"At a recent visit to the hospital, we saw several cases of intracranial tumour, or rather cases in which that diagnosis had been confidently made. One was that of a boy nearly 9 years of age, who was supposed to have a tumour of the middle lobe of the cerebellum. A case very like it, in the life history, and in which tumour of this part was found, is recorded by Dr. Hughlings Jackson in this Journal, November 4, 1871, p. 528. [See my former article.] In both cases there was, at the beginning, irregular gait; there were double optic neuritis and enlargement of the head. The patient we saw at our recent visit is in an advanced stage; he lies on his back with his legs and feet extended, and with his arms flexed acutely at the elbows. There is, however, no drawing back of the head; there is no arching of the back; and his abdomen, although sunken, is not at all hard. The head is almost constantly turned to the left, as are also the two eyes. From this position he never moves, except that the eyes occasionally wander to the right. [The following is a sketch of the cerebellar attitude in this patient's (1872) case.]

"His head is not only large—strikingly above the ears—but it is evidently enlarged, for the sagittal suture gapes widely (no relics of rickets in his body). A tumour of the middle lobe, by pressure on the veins of Galen, will cause hydrocephalus. Mr. Hilton thinks that the cause of the common form of hydrocephalus is the result of closure of the cerebrospinal aperture, which, according to him, is usually open. Possibly, then, in this case, the tumour closes this opening. But, whatever the mechanism may be, we see that, in an indirect manner, disease of the cerebellum can affect the cerebrum; there are mental symptoms in these cases. In a case closely like this in

CEREBELLAR PARALYSIS WITH RIGIDITY (THE RIGIDITY DUE TO CEREBRAL INFLUX).

("Cerebellar Attitude" in a Case of Tumour of the Middle Lobe of the Cerebellum, 1872).

Guy's Hospital, many years ago, Sir William Gull drew attention to the very general fading out of mind (a tumour of the middle lobe of the cerebellum was found, *post mortem*, as Sir William Gull had predicted). In the boy [1872] whom we saw there is great apathy; he never speaks unless he be spoken to, when he replies correctly. He lies on his back in one position all day, never taking notice of anything unless directly addressed. But against this is to be noted the fact that he can sing. Mr. Stephen Mackenzie has heard him sing several songs, which he had learned when well, and which he used to sing at home. He sang for the class, and apparently with great interest, the first verse of "Up in a Balloon."

In these cases, the intracranial pressure will be very gradually exerted (nervous matter bears slowly exerted pressure very well) and very evenly distributed—pressure by fluid. Thus we may account for the very general mental affection—the "fading out of mind." Power to sing is no very strong evidence of mental power. It is a largely automatic act. Aphasics can sometimes sing, and some idiots can. But it must be again remarked that the boy gives courteous and exact replies to simple questions; moreover, he replies readily. In Sir William Gull's patient, towards the end of life, the interval before replying was so long, that a hasty observer would have supposed that the patient was deaf or entirely demented; but at length the reply came, and it was slow and monotonous. The absence of deafness in these patients is noteworthy.[1]

"In the boy whom we saw there have been no tetanus-like fits, as there were in the case recorded in November [1871; see my former article]. From theoretical considera-

[1] In this footnote I give the next section of the 1872 report separately:—
"In cases of intracranial tumour it is as important to note the almost constant absence of deafness as it is to note the almost contant presence of blindness (partial or total)."
(1906.) That reference, 1872, is to defect of sight from optic neuritis; optic neuritis results from tumour in any part of the encephalon, and blindness from this pathological process is not owing to *destruction* of nervous structures by the tumour. I do not know of an "auditory neuritis," nor of an olfactory neuritis from intracranial tumour. I do not deny that there are these two kinds of neuritis; very likely there is an olfactory neuritis. Absolutely complete deafness of one ear in cases of tumour of a lateral lobe of the cerebellum is no doubt owing to squeezing of an auditory nerve.

tions Dr. Hughlings Jackson has been led to suppose that the changes which cause tetanus paroxysms [surgical tetanus] and the interparoxysmal 'set' of the trunk and limbs in [surgical] tetanus, are seated in the cerebellum, and not in the spinal cord, or not limited to it. But, since two such able observers as Lockhart Clarke and Clifford Allbutt have described changes in the cord in tetanus, he has refrained from fully stating this hypothesis. In the meantime, he diligently collects observations as to the tetanus-like affections, especially in the case where a gross (an obvious) lesion is likely to be found *post mortem*, to fix approximately the seat of the minute changes causing the paroxysm. Hence the interest of the tetanoid [tetanus-like] fits in the case related in November [1871]. In some cases of cerebellar disease there is only a fragment of the tetanus-like condition."

[The following (*British Medical Journal*, August 3, 1872, p. 125), is the sequel of the case mentioned in the preceding extract, from the Journal for July 20, 1872]:—

"In our reports, July 20, p. 67, is a note of several cases of intracranial tumour. We can complete the history of one of the cases, the first mentioned. The boy died July 24; and at the necropsy next day, a tumour of the size of a billiard ball (supposed to be a tubercle) was found in the middle lobe of the cerebellum, extending into the right lobe. The head, as mentioned in our last report [July 20, 1872], was very large; the parietal bones at the sagittal suture were found, *post mortem*, separated for more than half an inch; the cause of the enlargement of the head was hydrocephalus. Eighteen ounces of limpid fluid were obtained from the lateral ventricles; some was lost. There was no tubercle in any other part of the body. It should have been mentioned in our last report that the boy had strumous disease of the right foot and of one hand.

"For the reasons given with the notes of the case [1872], and in comments on a case strikingly like this one, which was published by Dr. Hughlings Jackson in this Journal, on November 4, 1871, the diagnosis of the position of the disease in these cases is not difficult. We must refer our readers to these comments."

ON DR. HUGHLINGS JACKSON'S VIEWS OF THE FUNCTIONS OF THE CEREBELLUM AS ILLUSTRATED BY RECENT RESEARCH.

THE HUGHLINGS JACKSON LECTURE, 1906.

BY SIR VICTOR HORSLEY, F.R.S., F.R.C.S., &c.

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THE Hughlings Jackson Lecture was founded in 1897, by the Neurological Society to do honour to Dr. Hughlings Jackson, and to acknowledge the great service he has done to humanity by his work on the functions of the nervous system.

The power of genius evinces itself by the happy use of analysis and synthesis, with as a result the discovery of principles. Dr. Hughlings Jackson's writings are *all* such expositions of principles, and no one rises from their perusal without realising that his conclusions are final without dogmatism, that a subject dealt with in his way always remains open for further scientific advancement, and finally that his reader has learnt far more than can be at once simply and adequately expressed. His teaching of first principles will always be gratefully remembered by the ever increasing army of neurologists, but this is the opportunity for those of us who have enjoyed the inestimable privilege of being immediately his pupils specifically to recall the profound depth of our indebtedness to him. We feel that his precept and example have influenced us both in methods of work and in methods of thought. Of the former I need only now particularise his constant inculcation of accuracy in observation, and, above all, his insistence on the necessity for correlating disturbances of function with the structural changes found after death.

Of even greater value has been his method of thought, by which a thinker can keep his mind open to appreciate broad principles even when it is most inclined to be narrowed by close attention to detail. Above all the intellectual width of his demonstration of the true principles of localisation of function in the central nervous system, and how with the co-existence of such localisation the functional activity of the nervous system as a whole can yet be estimated and philosophically considered.

Indeed the greatest principle which underlies all he has written, is the true nature of localisation of function in the central nervous system, namely, that it is relative and not absolute, that conceivably every part of the body is represented in every nerve centre, just as it of necessity is in the single primordial ovum.

And that, when the central nervous system considered functionally, is regarded in the view of Flourens as working as a whole the determination of action by any given part is never more than relative.

Perhaps Dr. Jackson's view of localisation in the central nervous system is not always sufficiently remembered and more especially when we are labouring with the difficulties of endeavouring to establish a differential diagnosis are we likely to forget that the whole machine is in active operation while our attention may be drawn to one part only.

In no sense is this truer than of the difference between and yet consonance of function in the cerebrum and cerebellum. Both organs are intimately correlated and both must be considered as invariably working together. Dr. Jackson's own confession of faith on this point is as follows (BRAIN, vol. x., p. 317): "I believe that the cerebrum represents all parts of the body, and that the cerebellum also represents[1] all parts of the body. . . . . That the cerebrum and cerebellum are engaged in any (every?) operation, will I suppose, not be doubted!!"

But it is not only the correlation between the cerebrum

[1] Perhaps as the term "representation" connotes in many minds the representation of movement more especially, it may not be out of place to remark that it equally includes the topographical presentation of all forms of sensation.

and cerebellum that is of importance, we have to add to these two organs a third, the equally important spinal cord.

Dr. Jackson has for thirty years [9] impressed upon us the necessity of always remembering that an interaction of these three parts is continually going on, that if one of them is put out of gear (*e.g.*, by an accidental lesion), then the functional energy of the remaining parts must assert itself. Let me here point out that this logical conclusion carries with it the further assumption that a certain degree of nerve energy is being constantly evoked, and as constantly is passing from one part to the other, and in that sense the central nervous system does work as a whole.

In confirmation of this the experimental observations of Gotch and myself in 1891, on the marked diminution in the resting electrical difference of the spinal cord when it is separated from the brain may be quoted [5], as well as the similar diminution or fall shortly before and at systemic death. Further, the "drainage" hypothesis of inhibition recently enunciated by Professor McDougall [12] also connotes the same idea, viz., that there is a certain quantum of nerve energy constantly being evoked in any given central nervous system; while finally, as I have often urged, the clinical pictures of surgical shock and of traumatic neurasthenia can only be explained on the same basis.

Thus each part of the nervous system influences another and for a long time Dr. Jackson has spoken of that share of energy emanating from the cerebrum or cerebellum (the latter more especially) as the *cerebral* and *cerebellar* influx respectively.

The evidence of its occurrence must, as he says, be a positive phenomenon, and thus the causation of (post-cerebral) rigidity and tonic contracture has been regarded by Dr. Jackson under the view that if the higher centres of the cerebrum or cerebellum are constantly exerting an (efflux) influence on the lower centres, it needs only to remove the higher organ, *e.g.*, the cerebrum, to allow of the lower, *e.g.*, the cerebellum, producing effects of its own unhampered. Hence the doctrine of *cerebellar influx* as

postulated by Dr. Jackson, but it is obvious that whatever be the ultimate explanation of early hemiplegic rigidity, his establishment of the principle of the production of motor phenomena of a chiefly tonic character by the lower level or bulbo-spinal apparatus when the highest level centres have lost their functional value, is of vital importance to neurology.

His in effect is the converse of Setschenow's original position [17] in respect of the influence of the corpora quadrigemina or optic lobes in the frog on the reflex function of the spinal cord.

The questions that rise immediately for discussion when such a principle is advanced are those which must govern its application, viz., enquiries into the specific or individual function of localised centres.

For the present occasion of this lecture we will select the cerebellum.

To begin and end with the cerebellum, it is partly necessary to allude to previous ideas of the action of that division of the neural axis in its relation to the cerebrum and the spinal cord.

Two hypothetical views thus prevail on the interaction between the cerebrum and cerebellum.

(1) The cerebellum favours the activity of the cerebrum.

(2) The cerebellum hinders the activity of the cerebrum.

I do not propose to enter upon a discussion of these views; because, at the present time, they deal with the dynamic activity of the cerebral and cerebellar centres only, and their output of energy, as estimated by the force of muscular contractions, wholly without reference to the facts of reciprocal innervation, as we now understand them, thanks chiefly to Sherrington's work [18], and with very little reference to the subdivisions of the cerebellar and bulbo-mesencephalic tracts and structures.

Any systematic attempt to analyse the cerebello-cerebro-spinal interaction, or to trace the paths and degrees of its influence, must be based on facts which can only be ascertained by the physiological investigation of the following tracts.

(I.) *Cerebellum Cortico-nuclear Paths.*

Cerebellar Cortex to nucleus dentatus.
,, ,, fastigii.
,, nuclei vestibulares.
,, pons varolii.

(II.) *Cerebellum Nucleo-Encephalic Paths.*

Nucleus dentatus to nucleus ruber.
,, ,, ,, thalamus.
,, ,, ,, griseum centrale tecti.
,, fastigii to nuclei nervi vestibularis.

(III.) *Cerebrum Thalamo-Cortical Paths.*

Thalamus to "excitor-motor" area.
,, "equilibial" area.

(IV.) *Cerebellum Nucleo-Spinal Paths.*

Nuclei nervi vestibularis to spinal nuclei.

(V.) *Side Paths of Correlated Importance.*

Rubro-spinal tract.
Tecto-spinal tract.

For the complete investigation of these tracts it will be ultimately necessary to trace to their original source the nerve impulses which pass along them. So far, the data to hand are very incomplete, but in order that we may, if possible, analyse Dr. Jackson's position, we can arrange the facts at our disposal, and we shall find that they justify certain positive conclusions, all of which are in gratifying harmony with his speculations.

The primary distinctions in functional or physiological value between them must be grouped as follows:—

(1) Cerebellar effects alone.
(2) Cerebellar effects plus cerebral influence.
(3) Cerebellar effects plus spinal cord influence.

Unfortunately these combinations of functions cannot be properly analysed by clinical research, and what we observe in any given patient are extremely compound effects. On the other hand, since, in accordance with the Jacksonian view of localisation of function, the seat of disease in the very large majority of cases acts as the "predominant part-

ner" among the co-excited portions of the central nervous system, the purposes of diagnosis are often sufficiently met.

Such a consideration brings us back to the point of view expressed on page 448 that there is reason to believe that the output or quantum of nerve energy is fairly constant for the same individual, assuming, of course, that his normal physiological health is maintained. It is, of course, an obviously necessary corollary to such a proposition, that, given a considerable output of nerve energy from one focus or centre (in this sense one organ) there must be so much less from the remainder of the nervous system.

In other words, any compound group of symptoms must, at some time, exhibit a predominant characteristic, and, if it can be detected, of necessity demonstrates the localisation of the disease as before said.

Though localisation can, by these means, be partly ascertained, it is through experimental investigation of the interacting encephalic organs in the lower animals alone possible to effect the differential analysis we need, for only in animals can the Galenical method of exclusion of parts be deliberately and restrictedly applied.

So far this has been thoroughly accomplished only between the excito-motor cortex cerebri on the one hand and the lowest level bulbo-spinal centres on the other.

This elemental analysis was begun by François-Frank and Pitres, and soon after confirmed by myself and others. Their well-known work consisted in the demonstration that whereas the muscle-contraction phenomena evoked by excitation of the cortex cerebri were essentially clonic in character, those obtained by stimulating the pyramidal fibres of the corona radiata conducting to the lowest level centres were tonic.

Precisely this condition had been previously surmised by Dr. Hughlings Jackson (*The Medical Examiner*, April 5, 1877) as follows:—"The cerebellum is the centre for continuous movements, and the cerebrum for changing movements. Borrowing medical terms to describe normal physiological conditions, the former (*i.e. cerebellar*) are tonic, the latter (*i.e., cerebral*) clonic."[1]

[1] The italics in brackets are mine.—V.H.

In all discussions by Dr. Jackson of the "cerebellar" influence as contrasted to "cerebral influence, his conclusions being drawn from clinical evidence, have been arrived at in full cognizance of the fact that all the groups of lower centres and paths, *i.e.*, cerebellar cortex, cerebellar nuclei intrinsic and extrinsic, as well as the bulbo-spinal system of lowest level centres are in action and co-operation.

The hind-brain in short, from the point of view of physiological topography, consists wholly of Dr. Jackson's lowest level[1] of representation, to which the cerebellum forming an appendage or "anhang" to the direct system is superadded. It is probably owing to the development in the higher mammalia of the specialised pyramidal projection system that the cortex cerebri has physiologically become so different from the numerous lower nerve centres in the mode of its discharge.

In saying this, however, it must not be forgotten that even this difference is not absolute, as indeed every instance of ankle clonus suggests, and Schäfer and I proved experimentally in 1885 that the outflow of every nerve centre is fundamentally a combination of tonus and clonus.

Precisely as with the representation of movement, so is the character of the outflow different according to the proportionate degree of one or other of these two features of tonus and clonus. Hence Dr. Jackson's assertion of the essentially tonic characteristics of the discharge of the lowest level centres, is not only of vital importance to cerebellar physiology, but it is in accord with the principles which underlie all nerve activity.

The best illustration that can be offered of the absolute truth of his general position on this point is that derived from a study of the epileptic convulsion. For this we happily need only to compare middle level and lowest level fits. In 1885-7 I found that in fits obtained by Magnan's absinthe method the type was clonic or not according to the proportionate activity of the cerebral cortex. Boyce,

[1] In Dr. Jackson's own words: "The lowest level extends, it is suggested, from the tuber cinereum to the conus medullaris. It is made up of sensory and motor centres of the cord, medulla, pons and aqueduct representing the body in detail."

at my suggestion, removed a cerebral hemisphere in cats, and proved that such extirpation of the highest cerebral influences resulted in fits of a strikingly tonic character. I proposed this year to Dr. Bouché, who has for some time been investigating at University College the details of the epileptic convulsion, that he should repeat these experiments to settle finally the question, and this he has done very completely. A good example is the following experiment which I witnessed with him. A cat, three weeks after removal of the left cerebral hemisphere, in excellent condition (*i.e.*, to superficial appearance normal, walking, &c., but exhibiting the typical atopognosis, mouvement de manège, &c., of an animal deprived of one cerebral hemisphere) was etherised, and the right (the remaining) hemisphere found to be normally excitable to electrical stimulation. A small dose of absinthe was injected into the jugular vein, and the typical result obtained of a tonic-clonic fit (chiefly flexor) on the left side of the body, but only a tonic (chiefly extensor) fit on the right. The attitude of the trunk and head was that of moderate flexion. While such a fit was in active development we made an instantaneous section of the mesencephalon. Immediately the clonic movements were changed for tonic in the whole body, the head was powerfully drawn backwards, the left limbs and trunk gradually sharing in the extensor movement.

This sudden conversion from (what remained of) the higher to the lower type of fit was very impressive, and constituted a remarkable demonstration of Dr. Jackson's dictum, that "Speaking very roughly and neglecting some parts of the body, the cerebellum represents movements of the skeletal muscles in the order trunk, leg, arm, preponderatingly extensorwise: the cerebrum represents movements of the same muscles in the order arm, leg, trunk, preponderatingly flexorwise." Dr. Bouché has established the constancy of this phenomenon by a series of successful ablations of one cerebral hemisphere and subsequent exploration with absinthe.

Perhaps the most brilliant experimental proof of Dr.

Jackson's position is Leonard Hill's series of experiments [7] in which he varied at will the clonic or tonic character of an absinthe fit by respectively leaving open or clamping the arterial supply to the cerebrum.

So, too, as regards the lowest level of all, viz., the spinal centres. The phenomenon first discovered by Schiff [16] and confirmed by myself and other workers, of extensor convulsions of the tail and hind limbs occurring even after section of the spinal cord is a clear illustration of the tonic-extensor character of a lowest level fit.

Finally, Loewenthal in an interesting work [10] on the effect of anæmia on the spinal cord obtained spinal fits in which with flexion of the spine and hips there occurred extension of the tail, knees and ankles.

An extreme example of such tonic response attributable in the main to cerebellar influence is illustrated in Dr. Jackson's communications published in 1871 and reprinted on pp. 431, 433, and 434 of the present number of this journal. The extreme contracture extension of the head (with lateral attitude in the fig. on p. 431) and lower limbs and tonic flexion of the fore limbs, are clearly demonstrated in the sketches by Sir Stephen Mackenzie, whose absence to-night we all regret. But we must postpone for a few moments the determination of the precise source of the representation of these movements.

With the guidance of Dr. Jackson's principles of localisation we must now endeavour to trace out the nerve impulses which have this power of evoking such a definite and specific association of muscular phenomena.

After what has been stated it follows that the direct investigation of the problem of Dr. Jackson's views on "cerebellar influx" naturally begins with the cerebellum itself.

As Thomas was probably the first (on anatomical grounds) to suggest [20], the cortex cerebelli and the (intrinsic) nuclei cerebelli must now be considered as two distinct organs.

Dr. R. H. Clarke and myself [3], by means of lesions *actually* restricted to the cortex cerebelli and tested by the

Marchi method have proved this opinion to be a neurological fact, and have demonstrated that :

(1) The cortex cerebelli sends no direct axones *via* the cerebellar peduncles to the brain or spinal cord.

(2) The cortical efferent axones terminate in the intrinsic nucleus dentatus, nucleus fastigii and nucleus emboliformis vel globosus.

(3) The intrinsic nuclei send efferent axones to the cerebral, spinal, and para-cerebellar, *i.e.*, bulbar nuclei.

The cytological investigations of Kölliker, Ramon y Cajal, &c., Retzius by the Golgi method, and Clarke and myself with polychrome blue, of these nerve centres, confirms the results obtained by the degeneration method, for whereas the cerebellar cortex is, histologically speaking, a structure *per se*,[1] the intrinsic nuclei are morphologically homologous with the efferent apparatus of the bulbo-spinal system. Particularly is this the case with the roof nucleus, one class of cells therein being typically spinal efferent in character.

So much for morphological evidence respecting the nature of the parts of the cerebellar apparatus. A phenomenon like that of tonic muscle contraction and the source of its maintenance, however, can only be investigated physiologically.

In 1904 I made a few experiments on dogs at Dr. Jackson's suggestion as follows :—In one group the mesencephalon was divided at the level of the intercollicular groove. The degree of the resulting decerebrate rigidity, *i.e.*, Dr. Jackson's "cerebellar influx" was then tested during and after removal of the cerebellar cortex in sections from above down. The hypertonus was not appreciably affected by such extirpations until the section involved the intrinsic and paracerebellar nuclei.[2]

In another group of but two experiments a large hori-

[1] The nearest morphological homologue to the Purkinje cell system is Clarke's column in the spinal cord and the cells of the "cerebral" root of the fifth nerve.

[2] In this connection it is important to remember that Schiff always contended that the earlier results of Flourens were correct, and that in the production of cerebellar phenomena of disturbance no effect was obtained until the lesions involved the "middle" of the white substance.

zontal lesion separating the dorsal half of the cerebellar cortex omitting the nuclei (save in one case where one of the nuclei fastigii was destroyed) was made three weeks before the division of the mesencephalon.

In these cases also no appreciable difference was observable in the decerebrate rigidity from that seen in normal animals.

Finally, this is the place to refer to the work accomplished by Thiele [19], in his investigation of the sources of the phenomena of hypertonus known since Sherrington's original investigation as decerebrate rigidity. Thiele found that apart from the autonomic action of the spinal cord, the plane of the principal source of the motor impulses evoking the rigidity is the level of the para-cerebellar nuclei, (*i.e.*, Deiter's nucleus, nuclei vestibularis, nucleus magnocellularis substantiæ recticularis, &c.).

So far, then, the evidence by experiments on disordered function tended to show that the immediate source of the "cerebellar influx" must be looked for in the basal or nuclear region of the cerebellar complex, and that the activity of the cortex cerebelli, though physiologically antecedent thereto, is not essential to the maintenance of the contracture phenomenon. The next step, naturally, was the direct re-exploration of the cerebellum, and that Dr. Clarke and myself have entered upon.

During nearly one hundred years various investigators have endeavoured to obtain evidence of cerebellar function by the excitation method,[1] either chemical, mechanical or electrical, but as a critical survey of the methods employed and of the results we have obtained up to the present show, it is probable that the effects observed by our predecessors were due to irritation of the subjacent nuclei and neigbouring tracts rather than to excitation of the cortex, except when the intensity of the stimulus was very high, that is, when the whole apparatus of cortex and nuclei is simultaneously thrown into an active state.

The differential exploration of the cerebellum entails among many other problems, first, that of focally irritating

[1] The Galenical method of investigation was, of course, in use for centuries before the excitation method.

any given point (square or cubic millimetre is the unit adopted by Clarke and myself), and secondly, of marking and destroying it for the purpose of accurate localisation, and of tracing secondary degenerations therefrom.

We[1] have, as described in a preliminary communication [3], been enabled to do this with a special apparatus carrying, by glass insulated fine needles, a current which can be readily changed from faradic to constant at will, to any desired spot in the encephalon.

This method of investigation has brought us to the following conclusions:—

First Conclusion.—*That the cortex cerebelli is inexcitable when compared to the cortex cerebri of the gyrus centralis anterior (ascending frontal gyrus).*

The immediate inference from this result is that the cortex cerebelli is not to be regarded as a motor centre or series of such centres, but as an afferent receptive centre from which axones pass to the ventrally placed efferent nuclei. In fact the cortex cerebelli is somewhat parallel in arrangement to the pallial visual cerebral centre.

It must now be shown how far this position has been already established by previous researches on other lines and methods.

The vermis region of the cortex cerebelli is the end station of the dorsal spino-cerebellar tract (of Flechsig) and the ventral spino-cerebellar tract (of Gowers). These paths are afferent tracts of the second order in the central neuronic system, and therefore are really homologous to the fillet. In that case the cerebellar cortex in the general scheme of the nervous system cannot be considered physiologically higher than the ventro-lateral region of the thalamus.

The termination of these spinal tracts of fibres in the cortex cerebelli discovered by Mott [13] deserves but has not yet received a minute numerical analysis. Mott traced the dorsal spino-cerebellar tract to the middle lobe (Elliott Smith) of the vermis and the ventro-lateral tract to the lobus centralis, while the further development of the nature

[1] Henceforth in the lecture wherever the expressions "we," "us," "our," are used they must be understood to refer to Dr. Clarke and myself.

and functions of these paths we owe to Tooth, Cohnstamm, Auerbach, and Sherrington.

I have myself made in different animals minutely limited lesions of both tracts, and like all other observers have fully confirmed Mott's original description. The direct cerebellar tracts have of recent years been made the subject of closer physiological study. Thus Marburg found that section of the tractus spino-cerebellaris dorsalis (Flechsigii) was followed by extension of the homolateral limbs which were abducted from the trunk and in a state of hypotonia although sensation and the spinal reflexes were both normal. So too Bing.

The time is not yet ripe to take advantage of the careful clinical work of Petrén on the ventral tract, but the general conclusions which can be justifiably drawn from the facts at our disposal, confirm Dr. Jackson's position that the atactic helplessness is primarily due to loss of information, *i.e.*, of afferent impulses.

As regards the question of the posterior columns being through their terminal bulbar nuclei partly a spino-cerebellar tract, it ought perhaps to be added here that like many observers, I have not traced any fibres into the inferior peduncle from the posterior columns.

This last named anatomical fact is but an extension of the physiological position arrived at by Professor Hermann Munk who demonstrated that section of the posterior columns did not produce ataxy whereas lesions of the posterior roots did.

Time unfortunately does not permit me to discuss further the enormously interesting and important work of Hering [6], Mott and Sherrington [14], Trendelenburg [21], and especially of Bickel [1], in this part, viz., the sensory or afferent side of the subject. I must strictly confine myself to the "motor" effects.

We recognise as the result of these morphological[1] and

[1] Previous to the determination of the existence of direct spino-cerebellar afferent paths, the existence of a relationship of the spinal cord areas to the middle lobe of the cerebellum had been theoretically suggested by Prideaux (*Medical Times and Gazette*, September 24, 1864. p. 340), from consideration of the relative evolution of: (1) tactual sensibility of the skin of the body (*i.e.*, spinal distribution) and of the lateral cerebellar lobes; of (2) muscular power and co-ordinated "muscular sense associated with development of the middle cerebellar lobe." I have to thank Dr. Hughlings Jackson for kindly referring me to Prideaux's paper.

physiological considerations, that the cortex cerebelli is an afferent recipient organ, as Edinger has always taught, and that the impressions from the trunk and limbs which it receives by these secondary neuronic systems from the spinal segments of the cord are of a special character, being already co-ordinated. What is true of the spinal afferent paths is apparently also true of the cranial nerve tracts, except that the intrinsic cerebellar and para-cerebellar nuclei additionally receive primary collaterals directly from the entering root fibres.

To return now to direct physiological investigation of the efferent phenomena. Clarke and myself have found that while excitation of the surface of the cerebellum is without effect, when on the contrary the insulated needles penetrate the cerebellar cortex, a minimal stimulus gradually becomes maximal as the needle points are made to approach the intrinsic nuclei, which are highly excitable, and give rise to such precise motor effects as to establish as follows our second conclusion.

SECOND CONCLUSION.—*That the intrinsic and the para-cerebellar nuclei are the efferent mechanisms of the cerebellum.*

To use Dr. Hughlings Jackson's terminology, these nuclei are the immediate source of the "cerebellar influx," the cortex cerebelli standing in the same causal relationship to the resultant motor effects, as does the afferent side of every nerve centre.

The muscular contractions which follow excitation of these nuclei must be surveyed from two points of view to compare them with Dr. Jackson's opinions; first, the topography of the part of the body represented, and secondly, the character of the muscular contraction observed.

(1) *Topographical Representation in the Cerebellar and Para-cerebellar Nuclei.*

As yet our researches are not completed, but provisionally it may be asserted that the essential motor representations of the parts of the body in the intrinsic and para-cerebellar

nuclei are in the former the movements of the eyes and head, in the latter more especially those of the trunk and limbs.

(2) *Character of the Muscular Contraction.*

The character of the motor effects obtained from excitation of the efferent side of the cerebellar apparatus is very striking and precisely of the kind originally ascribed by Dr. Jackson to it, viz., tonus and hypertonus. The movement even of the eyeballs is a steady swing over, which increases in contractile force as the excitation continues and without any clonic intermission of the motion or any development of any after-effect when the stimulation ceases. This being understood, the next questions respecting the character of movements derived from the efferent cerebellar mechanism are: (*a*) their relation to the middle plane of the body; (*b*) differentiation between flexion and extension.

(*a*) *Relation of the Cerebellar Influx to the Mid-plane.*—We find that when the stimulus is unilaterally applied to the intrinsic cerebellar nuclei, the effect as gauged by the action of the systemic muscles is homolateral, contrasting with the typical cerebral contra-lateral phenomena. A demonstrative example and proof of this principle which was first enunciated by Luciani [11], and especially by Risien Russell [15], is afforded by the result of our direct excitation of the upper part of the dentate nucleus alone, which we find evokes deviation of the eye of the same side, followed by conjugate deviation of both the eyes and head from the middle line to the same side.

So, too, excitation of the basal region of the dentate nucleus and the upper divisions of the para-cerebellar nuclei of one side evokes powerful bicipital flexion of the homolateral elbow.

(*b*) *Relation of Flexion to Extension.*—As regards the second point, Clarke and myself find, that while excitation of the basal region of the dentate nucleus with a maximal stimulus will produce, in addition to the homolateral conjugate deviation of the eyes and head, flexion of the homolateral elbow, a deeper excitation, *i.e.*, of the para-cerebellar nuclear region itself produces widespread and additional effects,

namely, extension of the contra-lateral elbow, hyperextension of the neck and trunk, with powerful extension[1] of the lower limbs. While therefore the forelimb is flexed at the elbow the hind limb is extended with the trunk.

Before pointing out the identity of these movements, we have observed, with those Dr. Jackson has taught for thirty-five years to be characteristic of the cerebellar apparatus and lowest level centres, I must revert for a moment to the debatable question of special representation in the central nervous system of the progression-like movement of extension of one limb associated with flexion of the corresponding contra-lateral limb, which is involved in the discussion of our observations on the para-cerebellar mechanisms.

The analysis of such regular and adjusted alternation of motor function carries with it several fundamental considerations, of which the most important is the first, namely, what is the highest plane in the neural axis from which such a characteristic phenomenon can be elicited by direct excitation?

Sherrington had in his work on decerebrate rigidity proved the existence of a co-ordinated organisation of the lowest (spinal) centres for the simultaneous performance of similar movements by alternated and contralateral limbs, and had proved the alternated character of representation in the spinal centres.

Thiele then, in an exhaustive enquiry by the excitation method combined with previous exclusion of the pyramidal tracts by degeneration, demonstrated that in the posterior third of the optic thalamus there exists a "centre" for progression, *i.e.*, walking.

The ablation experiments of various observers, especially Goltz, had proved for many years that, roughly speaking, the cerebral pallium was, in the quadrupedal animals at any rate, not absolutely essential to the functions of standing and walking.

[1] For a perfect illustration of this cerebellar movement in man, fig. 1, of Dr. Jackson's paper, p. 431. While such extension is observed to be the end-result, it is necessary to state that the ham-string and sural muscles are also in action, though with less efficiency than the extensors, which preponderate, to quote Dr. Jackson's words.

It follows, therefore, that the compound movement of walking is at least represented in the red nuclear region, and that one efferent path is therefore Monakow's or the rubro-spinal bundle.

Progression is, of course, an associated movement of primal importance and of co-ordination. It is, therefore, reasonable that it should have a parallel representation in the para-cerebellar nuclei, just as in the thalamus, and finally in the elementary manner of the reciprocal association of flexion and extension in the spinal segments.

We therefore consider the representation of the limbs in the para-cerebellar nuclei, as far as the upper limbs are concerned, to be compound and partly of the progression-movement type. But it must be further noted that in progression (for which the attitude of standing is practically requisite) the action of the remainder of the body, viz., the trunk and lower limbs, is especially that of extension. Thus the trunk is either held straight in extension or over-extended so as to cause a dorsal concavity while the lower limbs are extended as aforesaid; hence in man and the erect apes standing is impossible after destruction of the para-cerebellar mechanisms.

The "cerebellar influx" of the trunk and lower limbs is, therefore, *par excellence* extension, but for the minuter and proportionate representation of flexion and extension in the cerebellar and para-cerebellar nuclei reference must be made to a forthcoming paper by Dr. Clarke and myself.

Enough has now been said to show that Dr. Jackson's views of the nature of the cerebellar motor output, and his mode of interpreting the stimulation effects of gross cerebellar lesions have been amply substantiated by the findings of experimental differentiation, but his facts of illustrative interest must now be again quoted. The republication of his description of two cases on which his views have been in part founded makes this easy, and I would refer to papers 1 and 2 of the present number of this Journal.

It will be recognised that Dr. Jackson's conclusions were founded on instances of massive bilateral excitation of the whole cerebellar complex, and, therefore, in his cases

and Sir Stephen Mackenzie's striking sketches, we see that the features of the alternate movements of progression are entirely overwhelmed by the cerebellar disturbance with as resultant a strikingly peculiar position of the body, viz., hyper-extension of the head, trunk, and hind limbs associated with powerful flexion of both elbows.

I think it must be granted that, under the circumstance of secondary intraventricular distension occurring in such cases the removal of the cerebral influx is a natural consequence of the condition. This special combination of muscular contractions, which Dr. Jackson has termed "the complementary inverse of epileptiform seizures," is comparatively rare, but as a contribution to the subject I may allude to an instance of glioma cerebelli in a boy whose case I recently reported to the Society. In that case we repeatedly observed severe "cerebellar fits" of Dr. Jackson's type, and an operation enabled a glioma of considerable size to be removed from the right lateral lobe of the cerebellum. The boy remains in perfect health, and it can only be assumed that the para-cerebellar structures were at least severely pressed upon only and not destroyed. So, too, in Dr. Farquhar Buzzard's very interesting case of diagnosed lesion of the cerebellum, a like affection of the nuclei doubtless existed. The discussion of the subject of extension as Dr. Jackson's characteristic muscular movement resulting from cerebellar and lower level centres active influence cannot be closed without allusion to his teaching concerning the representation of eye movements in the cerebellum. From the first Dr. Jackson has considered that since the cerebellum is the essential organ in locomotion and progression as just narrated, there must be represented therein associated ocular movements.

Now, in accordance with this view, Dr. Clarke and I have found that the eye movements described by Ferrier as obtainable by excitation from the surface of the cerebellum, owe their motor genesis, not to localised centres in the cortex cerebelli, but to foci in the intrinsic nuclei.

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nucleus, and consists of a conjugate deviation to the homolateral side, and further, that as the excitation point approaches the midline (and the neighbourhood of the nucleus of the sixth nerve below), differences in deviation tend to appear.

The analysis of these functions will take some time, probably years, to complete, but one condition is well worthy of notice, namely, that originally observed by Magendie, which Dr. Jackson has spoken of as skew deviation, and in which the eyes are directed in opposite ways. Thus one visual axis is directed up and in, while the other is down and out.

Dr. Jackson has always urged that this apparently paradoxical phenomenon is really only half a normal binocular conjugate deviation, and he traces skew deviation to a destructive lesion affecting one half of an individual's apparatus for the locomotory appreciation of distance and position in space, the localisation of such an apparatus being as yet incomplete, but unquestionably in the temporo-cerebellar complex. The experimental illustration of the truth of this view occurred by the following happy accident.

Having for other purposes selected the armadillo (*Dasypus villosus*) as almost the lowest available mammalian type of cerebellum, Dr. Clarke and I were gratified to find on excitation of the intrinsic nuclei that skew deviation was the rule in this animal,[1] and that inasmuch as it is remarkably monocular in almost every position of its head, it clearly illustrates Dr. Jackson's views most completely, since it normally employs but one half of a binocular apparatus.

The coaptation of eye movements with those of the limbs in locomotion of the body by means of the limbs is, theoretically a fundamental physiological fact in man, and the higher animals. Dr. Clarke and I suggest, therefore, that the true results of cerebellar excitation are those which give first place to the representation movements of the eye rather than of the limbs, which conclusion is in general accord with Ferrier's pioneering observations.

The *antecedence* of eye movements in all skeletal mus-

[1] The three animals, monkey, dog, armadillo, thus arranged, show very strikingly the increase in skew deviation as the vision becomes less binocular.

cular actions is well recognised in most cases, but the fundamental importance of such time relation in cerebellar physiology has scarcely been so much appreciated.

Conclusion.

This brings me to the general conclusion with which any statements of cerebellar function must now be summarised, viz., that all research, ancient and modern, tends to confirm the view of Flourens, Luciani, Jackson, and Edinger, that the cerebellar cortex is the first chief station of representation of the afferent basis of movements of all the skeletal muscles.

Gratefully to acknowledge to Dr. Jackson his share in bringing order out of confusion in this and many other departments of neurology is the object of our meeting here; and greatly as I feel the honour of being selected as the Society's representative to-day, I esteem the charge of communicating your thanks to him as my highest privilege.

May I add the expression of our hope that we may long continue to enjoy his illuminating speech and his stimulating example.

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ON THE PROPRIO-CEPTIVE SYSTEM, ESPECIALLY IN ITS REFLEX ASPECT.

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REFLEXES, as judged by various criteria, are variously classified. Each arrangement, in so far as based on distinctions truly broad, may be of service to neurology. For reflexes have, besides intrinsic practical importance, a value for theory. Among them may be expected prototypes which, in simple form, represent generic divisions of nervous reaction in general. Any natural classification of them may therefore assist recognition of groups of nervous reactions embracing those even of high complexity.

In every reflex a decisive stage is that initial one wherein are started the centripetal impulses, which, in due course, excite the central reaction. In natural reflexes these initiatory impulses are excited by the reaction of peripheral apparatus, itself excited by some adequate stimulus. In so far as sensation accrues to the central reaction, the peripheral receptive apparatus may be termed sensory. But sensation may not accrue, although a central reaction is set up. In many cases it is hazardous, and for many purposes it is immaterial, to state whether sensation does or does not attend the central reaction; in such cases the peripheral apparatus is preferably so designated as neither to predicate nor to preclude sensation as being one of the features of the central reaction. Adopting the term *receptor* as a neutral one to apply to the peripheral apparatus for this purpose, it is obvious that, in trying to analyse the functions of the central nervous system, recourse may be had to the qualities of the receptors as furnishing a criterion. On that basis various parts of the central nervous system may have, and have

already had, their functions distinguished. Certain inferences as to the general scope and relationships of reflexes may be drawn from the broad scheme of distribution and situation of the receptors in the body.

The receptor organs, if one regards their distribution from a broad point of view, fall naturally into two great groups, as judged by their locus in the body. They are distributed in two great separate fields, each field circumstanced fundamentally differently from the other. Multicellular animals, regarded broadly throughout a vast range of animal forms, are cellular masses which present to the environment a surface sheet of cells and, underneath that surface sheet, a bulk made up of cells more or less screened from the environment by the surface sheet overlying them. The surface sheet is directly exposed to the environment, and is adapted to react to many of the factors composing that environment, these factors constituting stimuli. Bedded in the surface sheet are numbers of receptor cells, developed in adaptation to the stimuli delivered by environmental agencies. Many of the agencies by which the environment acts on the organism do not, however, penetrate to the mass of cells forming the organism's deeper parts. Thus various stimuli in the forms of light and heat, and localised pressures, and chemical substances expend themselves as stimuli at the surface sheet, and do not penetrate into the depth of the organism. But the deep tissues, although devoid of receptors adapted to these surface-reaching stimuli, are, nevertheless, not unprovided with receptors. They have receptors of other kinds apparently specific to them. Some agencies act not only at the surface of the organism, but also through its mass. For some of these agencies no receptors appear to be adapted; for instance, there seem to be none adapted for the Röntgen rays. For others of more common occurrence, receptors in the deep tissues seem to have been evolved. The deep receptors appear to be very usually adapted to mechanical stimuli of certain kinds. Thus, they seem adapted to react to the compressions and strains produced by muscles, and an important adequate agent for them seems to be mass acting in the

modes of weight and inertia, involving mechanical pressures and mechanical stresses.

The main fields of distribution of the receptor organs fundamentally distinguishable seem, therefore, to be two, namely, a *surface* field constituted by the surface layer of the organism, and a *deep* field constituted by the tissues of the organism beneath the surface sheet.

But the surface field is further broadly subdivisible. Its subdivisions are two. Of these one lies freely open to the numberless vicissitudes and agencies of the environment; it is co-extensive with the so-called external surface of the animal. It is cutaneous in the widest sense of that term. It possesses as receptive organs not only those of touch, &c., in the skin proper, but also the eye, nose, and organ of hearing. This subdivision of the surface field contrasts with a second subdivision of it, constituted by what is commonly termed the internal surface of the animal, the alimentary or intestinal surface. This latter surface is, it is true, in contact with the environment; but the environment with which it is in contact is a portion of the environment greatly modified from the general environment outside by lying almost completely surrounded by the animal itself. This part of the receptive field of the animal's surface, which is turned inward upon the alimentary contents, may be termed the *intero-ceptive*, in contradistinction to that larger part of the surface field which looks outward upon the free environment in general, and the latter may from that circumstance be termed the animal's *extero-ceptive* surface.

It is this latter, the *extero-ceptive* field, which is most rich in receptive organs, both as regards number and variety. For this to be the case, is in accord with what might be expected. It is this *extero-ceptive* field which, facing outward on the general environment, receives and has received for countless ages the full stream of all the varied agencies for ever pouring on it freely from the outside world. Mere enumeration of the different species of receptor organs recognisable in it, suffices to illustrate the importance of this great field in regard to "reception." It contains receptors specifically adapted to react to mechanical contact, to cold,

to warmth, to light and sound, and agents, whether mechanical, chemical, or radiant, which inflict injury (*noxa*). A number of these species of receptors are distributed to the extero-ceptive field exclusively; receptors adapted as these are not known to exist either in the deep field or in that part of the surface field termed *intero-ceptive*.

Less, however, is known concerning the receptors of the intero-ceptive field. Of certain points regarding them we may feel fairly assured. The portion of body surface—the so-called "internal"—which forms their field of distribution, is alimentary and digestive in function. For purposes of retaining food, digesting and absorbing it, an arrangement of common occurrence in animal forms is the deep recessment of a part of the free surface. In this recess a fraction of the environment is more or less surrounded by the organism itself. Into the sequestered nook so formed the organism by appropriate reactions gathers morsels of environmental material, whence by chemical action and by absorption it draws nutriment. Hence that surface of the organism which lines this recess is in contact with a part of the environment largely separated from, and profoundly modified from, the environment in general. This specialised part of the environment is specially rich in digestive stimuli, and by the screening and selective action of the organism itself is practically free from many of those stimuli which abound in the general environment at work on the creature's outer surface.

The receptive field lining this digestive chamber, this kitchen, appears especially adapted to chemical stimuli. At the ingress to the chamber, namely, in the mouth, species of receptor organs occur, whose adequate stimuli are chemical. The sensations induced through them are gustatory. Their reflex actions include reflex secretion of saliva. But further inward, along the digestive surface, we know as yet comparatively little about the receptor organs and the reactions they evoke. The fine work by Bayliss and Starling has shown that the pancreas can be excited to secrete by chemical agency without direct intervention of the nervous system at all. From a number of facts it seems clear that

the internal digestive surface of the body is relatively sparsely endowed with receptor organs, as compared with the free extero-ceptive surface outside. The number of afferent nerve fibres in the sympathetic system, which is mainly a visceral system, is small in comparison with the number of afferent fibres in the cerebro-spinal nerves from the skin and receptive structures of the external surface. Recent observations by Warrington show that the number of cells of the spinal ganglia which exhibit regressive changes after severance of the white rami—containing the afferent fibres from viscera—forms but a small fraction of the total number of the cells in those ganglia. Similarly no *wholly* afferent nerve-trunk appears to exist in the sympathetic system, but such are common in the nervous system belonging to the extero-ceptive surface. Conversely, in the latter system, no *wholly* efferent nerve-trunk is known, though in the sympathetic such trunks exist.

Returning to the receptor organs bedded in the deep tissues, and to the study of the reactions which they subserve, two characters attract attention as differentiating them from those of the surface field, whether extero-ceptive or intero-ceptive. Of these features, one is that the *stimuli* effective on the receptors of the deep field differ fundamentally from those operative on the receptors of either subdivision of the surface field. Most of the stimuli by which the external world commonly acts on the surface of the animal are excluded from the deep field. But the organism itself, like the external world surrounding it, is a field of ceaseless change where internal energy is continually being liberated, whence chemical, thermal, mechanical, and electrical effects appear. It is a microcosm in which forces which can act as stimuli are at work, as in the macrocosm around. The receptors which lie in the deep tissues appear adapted for excitation by changes going forward in the organism itself. These changes work, it appears, largely through the agency of mass with its mechanical consequences of weight and inertia, and also largely through mechanical strains and alterations of pressure resulting from contractions and relaxations of muscles. *There-*

fore, a character of the stimulations occurring in this deep field is that the stimuli are traceable to actions of the organism itself, and are so in much greater measure than are the stimulations of the surface field of the organism. Since in the deep field the stimuli to the receptors are delivered by the organism *itself*, the deep receptors may be termed *proprio-ceptors*, and the deep field a field of proprioception.

But the bodily reactions which act as stimuli to the deep receptors are often themselves directly excited by external stimuli delivered at the animal's free surface by the outside world. Thus, the mechanical strain or compression which stimulates some deep receptor arises, as the result of some antecedent reflex movement provoked through some surface receptor, usually of the extero-ceptive field. The immediate stimulus for the reflex started at the deep receptor is supplied by some part of the organism itself acting as agent; but that part became an agent of stimulation in the majority of cases because of a reaction induced in the deep organs of the animal by some environmental agent acting on receptors of the animal's surface. *Hence the reactions produced by the receptor organs of the deep field are results primarily due to the stimulation of the organism by itself, but secondarily due to the stimulation of the organism by the environment.* This secondary relation to surface stimuli is habitual. Reflexes arising from proprio-ceptive organs come, therefore, to be habitually attached and appended to certain reflexes excited by surface receptors. In a muscular movement induced by a stimulus to the skin of the spinal dog, the change in form and tension of the muscles, the movements of the joints, &c., excite receptors in these tissues and their accessory structures, and these proprio-ceptors in their turn excite a reflex in their own arcs. A character of the reflexes of the proprio-ceptive field is therefore that in contradistinction from those of the surface fields, they are related not primarily, but only secondarily to the agencies of the environment.

It would appear that in many cases the proprio-ceptive reflex which thus ensues as a secondary reaction, *allies itself*

in its effect to the primary reflex excited from the extero-ceptive surface, and reinforces it. The flexion reflex of the hind limb of the dog can be excited by a stimulus applied to the skin of the foot. A similar flexion reflex of the limb can be excited by stimulation of the afferent nerve-fibres of the flexor muscles themselves. A subliminal stimulation of the skin of the foot becomes effective when a subliminal stimulation of the afferent nerve of one of the flexor muscles is delivered synchronously. These stimuli mutually reinforce each other. The reflex due to the extero-ceptive surface is reinforced by the appropriately chosen proprio-ceptive reflex. On the other hand, a flexion reflex of the limb provoked from the skin can be cut short by reflex inhibition on stimulating the afferent nerve of a flexor muscle of the opposite limb. In this case the second or proprio-ceptive reflex restores the posture of the limb which the extero-ceptive reflex had disturbed. In this case the proprio-ceptors evoke a *compensatory reflex* in the opposite direction to the reflex excited from the skin, the extero-ceptive surface.

A third character of the receptors of the deep or proprio-ceptive field seems their tendency to induce and maintain *tonic* reactions in the skeletal musculature. The steady, long-continued, untiring, and, for the most part, mild contraction, which is observable so frequently in skeletal muscles, and is known as their tone, where traceable to spinal reaction, has been traceable reflexly not to the receptors of the surface, but to the proprio-ceptors of the muscles and their accessory structures. Thus, the proprio-ceptors of the limbs appear to be a main source of the reflex tonus of the muscles of the limb. In the decerebrate dog, the tonic extensor rigidity of the leg seems reflexly maintained by afferent neurones reaching the cord from the deep structures of the leg itself; for after severance of the afferent roots of the nerves of the limb the tonic rigidity hardly appears, although it does appear unimpaired when the main skin nerves have been severed. Similarly, if in the spinal animal the knee-jerk be accepted as evidence of a spinal tonus in the extensor muscle of the knee, this tonus seems maintained by afferent fibres coming from the exten-

sor muscle itself, since the knee-jerk is extinguished by severance of those fibres. Again, the rapidity of onset of *rigor mortis* in a muscle is high in proportion as its tonus, prior to death, has been high. Section of the afferent roots of the nerves of the limb, prior to death, not only lowers the normal tenseness of the muscles of the limb, but also delays in them the onset of *rigor mortis* as judged by the onset of *vost-mortem* stiffness at the knee; and this delay is not observable when the skin nerves only have been severed.

It is often asked, What may be the utility of the tonus of skeletal muscles? A suggestion may be hazarded, which is germane to the present argument. One function this tonus may serve is that of an adjuvant to so-called muscular sense. The steady, mild tension which the muscles exert in virtue of their reflex tonus may, by acting on joint surfaces, tendons, and muscles, &c., assist the functioning of the sense organs belonging to those structures, much as the reflex tonus of the constrictor pupillæ is adjuvant to the visual receptive surface, and affords the eyeball a favourable condition for its function. But the reflex tonus of skeletal muscle still persists, after destruction of the higher centres to which we have to relegate the operations of sense, and *inter alia* those of the so-called "muscular sense." And it is clear that the receptors of muscles, joints, &c., are concerned with much outside the production of sensations. The reflex tonus of skeletal muscles must therefore have, it would seem, a purpose outside that of directly subserving muscular sense. I would suggest that this reflex tonus is the expression of a *neural discharge concerned with the maintenance of attitude.* Though of many reflex reactions the outcome is movement and the muscles reacting are thus used as organs of motion, much of the reflex reaction expressed by the skeletal musculature is not motile, but postural, and has as its result not a movement, but the steady maintenance of an attitude. The bony and other levers of the body are thus held in certain positions, both in relation to the horizon and to one another. The frog, after removal of its cerebral hemispheres, rests squatting in its tank preserving an attitude very different from that which gravi-

tation would give it were its musculature not in action. Evidently a great part of its skeletal musculature is steadily active all the while, antagonising gravity in maintaining the trunk semi-erect, the fore limbs semi-extended, and the hind limbs tautly flexed. Innervation and co-ordination are as fully demanded for the maintenance of a posture as for the execution of a movement.

A posture of the animal as a whole, a *total* posture, is a complex built up of postures of portions of the animal; segmental postures, just as movement executed by the animal as a whole, *total* movement, *e.g.*, locomotion, is compounded of segmental movements. With only the hinder portion of its spinal cord intact, the hind limbs of the frog still maintain a posture. Owing to spinal reflex action, the hind limbs are then kept flexed at hip, knee, and ankle. When displaced from that posture they return to it. The deep receptors of the limbs, the proprio-ceptors, give to their segments, namely, those of the hind limbs, by local spinal reflex action a definite *attitude*.

The vertebrate animal, as regards the scheme of arrangement of its spinal and cranial reflex arcs and the musculature which they innervate, may be regarded as a fore and aft series of segments. In each segment there tend to recur reflex arcs of similar functional kind to those occurring in the other segments. For instance, to each segment there belong reflex arcs arising at the skin surface (extero-ceptive) and arcs arising in joints, muscles, tendons, &c. (proprio-ceptive). The arcs of analogous function belonging to successive segments unite to a homogeneous reflex system extending more or less continuously through the length of the animal. Thus it is that the *proprio-ceptors and their reflex arcs have, in their sum total, to be treated as a proprio-ceptive* SYSTEM.

As the segments, *e.g.*, of a vertebrate, are followed toward the head end of the animal, that is, toward the end which leads in habitual locomotion, the receptor organs are found to exhibit in the head or leading segments a greater development and general importance than in the other segments. This is strikingly obvious in the receptors of the external

surface. It is at the leading end of the animal that the great extero-ceptors constituting the eye and the organs of hearing and of smell are met. This leading end it is which, when the creature moves in the direction of its habitual locomotion, explores that portion of the environment into which the body is about to pass. And when the creature is not itself in active locomotion, but, as often happens, allows a moving environment, *e.g.*, a stream of water or a current of air to drift by, the posture assumed by it is usually such as makes the moving environment impinge upon the head segments first. It is not surprising, therefore, that the reflex arcs and centres of the head or leading segments are found to dominate the arcs of analogous function in all the segments behind their own. A segmental series of reflex arcs of analogous function in its integration to a unified system tends to exhibit a functional hierarchy, in which the reflex arcs belonging to the head are supreme. As regards the system of the proprio-ceptive arcs, it remains to enquire whether there also the head segments are of higher general importance than the rest, and exert a special predominance in their system.

Taking the case of the vertebrate, in it there lies in one of the leading segments (head segments) a receptor organ, the *labyrinth*, recessed off from the surface as a sort of cyst. This receptive organ is adapted to mechanical stimuli. It consists of two parts, both endowed with low receptive threshold, and with refined selective ability to differentiate stimuli. One part, the otolith organ, is adapted to react to changes in the incidence and degree of pressure exerted on its nerve-endings, by contents of higher specific gravity than the fluid otherwise filling the organ. The second part, the semi-circular canals, appear to react to minute mass movements of fluid contained within it. These two parts may be taken as typically constituting the labyrinth. The incidence and degree of pressure of the otoliths upon their receptive bed alter with changes in the position of the segment, in which the labyrinth lies, relatively to the horizon line. Similarly, movements of the segment stimulate the labyrinthine receptors through the inertia of the

labyrinthine fluid as well as of the otoliths. There are some obvious resemblances between these labyrinthine functions and the functioning of the proprio-ceptors, *e.g.*, of a limb. In the stimulation of the labyrinth, the active agent which directly excites the receptor is a portion of the organism itself, and is not an item of the environment. Moreover, the stimulation is provoked commonly by a reactive movement of the organism itself, and that reactive movement is commonly in its turn a response to an environmental stimulus acting on some receptor of the extero-ceptive surface. Both these are points of resemblance to the conditions above shown to obtain in regard to the stimulation of the proprio-ceptors, *e.g.*, of a limb. The segment to which the labyrinth belongs is commonly rigidly conjoined with the other segments composing the head. Postures and movements of the head are thus the immediate causes which stimulate the labyrinth. Thus, the labyrinthine receptors, like the proprio-ceptors in other segments, are stimulated by the animal itself as agent, though secondarily to action of the environment at work upon the animal.

There is another functional likeness between the labyrinth and proprio-ceptors elsewhere. The latter, as was mentioned, are the source of a reflex tonus in the skeletal muscles. J. R. Ewald and others have shown that to the labyrinth is traceable a share of the neural tonus of many muscles. Ewald concludes his work on the labyrinth by designating it the tonus labyrinth. And these tonic actions of the proprio-ceptors and of the labyrinth appear to reinforce mutually. Thus the tonus of the extensor muscle of the knee, in the cat and dog, appears to have, under many circumstances, a combined source in the proprio-ceptors of the limb and in the receptors of the labyrinth.

A further functional resemblance between the labyrinth and other proprio-ceptive organs, lies in both of them initiating compensatory reflexes. It was mentioned that to the proprio-ceptors of the hind limb seems traceable in the spinal animal, the active reflex resumption of a posture temporarily disturbed by an intercurrent movement. The

limb is returned to its original attitude by help of a movement which is reflex and traceable to afferent nerves from the articulo-muscular mechanisms of the disturbed part. Similarly, the reflex posture and the compensatory reflexes of the head are largely traceable to the receptors of the labyrinth. From the labyrinth are excited reflexes which adjust its segment—and with that segment the others of the head are rigidly conjoined—to the horizon line. That the adjustment is to the horizon line seems particularly shown by the eyeballs. The retinæ, those refined photo-receptive patches of the head surface, conduct reflexes delicately differential in regard to space. The due reaction of the animal to stimuli, acting above or below, or to right or to left, in the photo-receptive patches, depends on a fairly constant relation being kept between the normals of the patches and true horizontality. This is the meaning of the laws of Donders and Listing regulating eye-ball movements. The photo-receptive patches are set movably in the head. By the action of muscles they can retain their bearing to the horizon, although the head itself shifts its bearing to the horizon. The reflex control of these muscles lies largely in the power of impulses from the labyrinth. When temporary causes disturb the usual relation of the normal of the head to the normals of the retinæ, compensatory reflexes bring back the parts to their previous habitual relation both to each other and to the horizon line. These compensatory reflexes, as well as the habitual maintenance of that primary harmonious attitude, are largely traceable to the operation of the reflex arcs which arise in the receptors of the labyrinth. Thus, *as the proprio-ceptors of a limb are in large measure responsible for the reflex posture and the compensatory reflexes of the limb, so the labyrinthine proprio-ceptors are largely responsible for the reflex posture and the compensatory reflexes of the head.*

It seems therefore legitimate to include the labyrinth within a great and distinctive system, reflex and sensual, which from the special conditions attaching to the stimulation of its receptors, may be termed a system of proprioception. This *proprio-ceptive* system possesses, in virtue of

the labyrinth, an important component placed in the head segments. With other receptive systems composed from segments in series, the receptors of the head segments tend functionally to dominate the rest. It is so, likewise, in the proprio-ceptive system. The influence of the proprio-ceptors of a limb on the reflex maintenance of posture is confined practically to the limb; the influence of the proprio-ceptive organ of the head upon posture is exhibited not only by the posture of the head itself, including the eyeballs, but extends to the limbs and trunk as well. Thus, destruction of the labyrinth in fish, frog, pigeon, dog, produces not only malposture of the eyeball and head, but of the limbs and body as a whole. In man the "knock-out" blow, where the lower jaw conveys concussion to the labyrinth, reduces in a moment a vigorous athlete to an unstrung bulk of flesh, whose weight alone determines its attitude, if indeed a reactionless mass can be described as possessing attitude at all. The proprio-ceptor of the head, namely, the labyrinth, embraces within its field of reflex effect all of the segments of the body. The posture of the head, guided as that is by the labyrinth, is itself dependent on the action of muscles in a whole series of segments behind it, and conversely the posturing of the entire animal often has as its chief objective the bringing of the head into some requisite direction. Hence the cranial proprio-ceptive organ is in reflex touch with a whole train of the segments of the body, and these latter may be regarded as in a measure appended to the otic segment which contains the cranial proprio-ceptor.

As mentioned above, a posture of the animal as a whole, a total posture, is composite in the sense that it is built up of postures of portions of the animal, segmental postures. The postural flexion of the hind limbs of the spinal frog is maintained by spinal reflex action. It is maintained when the animal is inverted, and rolled over upon its back. A frog, after removal of its fore and mid-brain, when inverted, reverts at once by reflex action to the right-side-up posture. It corrects the malposition as does a normal frog. But when the labyrinths or their nerves are destroyed, this compensatory reflex is at once lost. The reflex evoked by

the cranial proprio-ceptor keeps the world right side up for the animal, by keeping the animal right side up to the world. The proprio-ceptors of the spinal region are unable to do this. The proprio-ceptors of the limbs give to the limbs a definite reflex attitude less in reference to the external world than in reference *to the other segments of the body, i.e.,* to the rest of the body. The proprio-ceptive organ of the head, on the other hand, gives the animal a definite reflex attitude in reference *to the external world.* Thus, it maintains not merely a limb in flexion or extension, but a posture of the whole animal in regard to gravitation.

Evidently the proprio-ceptive organs of the leading segments play in the proprio-ceptive system a *rôle* which is a dominant one. The reflex arcs of the proprio-ceptors of these segments embrace peculiarly large fields of the skeletal musculature. The reflex arcs of the proprio-ceptors of the other segments act only on more limited fields of musculature. In conformity with this, the proprio-ceptive organs of the leading segments possess long internuncial efferent paths; for instance, the vestibulo-spinal proceeding from Deiter's nucleus to the eye nuclei, and to all levels of the spinal cord.

It was seen that the proprio-ceptive system can be distinguished as a functional unity extending throughout the whole series of segments of the vertebrate organism. Other functional reflex systems are similarly distinguishable as segmentally composed. In all such systems a tendency is manifest for the nervous centres of the system to fall under the dominance of one of their own number, situate so as to receive the afferent paths from a pre-eminently important group of the receptors of the system. The whole reflex mechanism of the system tends to be, so to say, focused upon the centre belonging to that group. Thus, in the reflex system dealing with respiratory movement, the chief centre controlling the taxis of that movement lies in the bulb, where afferent paths from the lung itself enter and make central connections. In the proprio-ceptive reflex system, the most important group of receptors was seen above to be the labyrinthine. Concordantly with this the

central mechanism of the proprio-ceptive system is built preponderantly and developed chiefly in the hind brain, which receives the vestibular nerves. Johannes Müller, years ago, declared the tuberculum acusticum, with its fibres received from the labyrinth, to be in Petromyzon the representative of the cerebellum of higher fishes. Johnston recently insists on the close structural resemblance and connection between the tuberculum acusticum and the cerebellum in this same low aquatic vertebrate. To the region of the hind brain, which receives the vestibular nerves, internuncial paths from many proprio-ceptive centres in other segments of the nervous axis converge as to a focus. These, with those from the labyrinth, form there a sort of organ of confluence for them all. A central organ of high complexity is thus formed in this region. Its size, as traced from animal species to animal species, strikingly accords with the range and complexity of the habitual movements of the species; in other words, with the range and complexity of the reflex taxis of the skeletal musculature. This central organ is the *cerebellum*.

The symptoms produced by injury or destruction of the cerebellum, in whole or in part, resemble, therefore, in various respects the disturbances set up by injury of the labyrinth itself, and in some respects the defects produced by damage of the proprio-ceptors of the body regions. The cerebellum likewise influences the tonus of skeletal muscles as would be expected, seeing the influence on that tonus exerted by proprio-ceptive arcs in general. There is a close connection structurally and functionally between the cerebellum and the so-called motor region of the cerebral hemisphere, just as there exists an intimate association of the simpler proprio-ceptive arcs and reflexes to the mechanisms of extero-ceptive reactions. Knowledge on many points is not yet sufficient to warrant an attempt to offer an adequate definition of the function of the cerebellum. Many authorities have defined it as the centre for the maintenance of the mechanical equilibrium of the body. Others regard it as the organ for the co-ordination of volitional movement. Herbert Spencer suggested that it is the organ of co-ordina-

tion of bodily actions in regard to space; the cerebrum, he suggested, being the organ of co-ordination of bodily actions in respect of time. Hughlings Jackson, nearly thirty years ago, insisting on his co-operative antagonism between the cerebellum and the cerebrum, wrote with characteristic penetration of thought, "The cerebellum is the centre for continuous movements, and the cerebrum for changing movements." Lewandowski considers the cerebellum the central organ of the muscular sense. The function attributed to it by Munk is the finer taxis of the maintenance of bodily equilibrium. And Munk well points out that its influence upon certain muscles and their actions is much greater than it is upon others. Luciani, the most widely-recognised physiological authority on the cerebellum, describes it as the organ which, by unconscious processes, exerts a continual reinforcing action on the activity of all other cerebro-spinal nerve centres.

All these separate pronouncements appear to me to harmonise with the supposition that this organ is the chief co-ordinative centre, or rather group of centres, of the reflex system of the proprio-ceptors. *The cerebellum*, it would thus be permissible to describe as *the head-ganglion of the proprio-ceptive system;* and in the proprio-ceptive system, as in other systems of receptors and their arcs, the head-ganglion is the *main* ganglion.

The above brief sketch has allowed of no treatment of the proprio-ceptors as generators and modifiers of sensation. The system, whose basis they form, is, however, no less distinguishable, when regarded in its sensual aspect, than when exhibited, as in the present condensed statement, in the light only of its reflex action upon the musculature.

A PRELIMINARY NOTE ON THE COMPARATIVE ANATOMY OF THE CEREBELLUM.

BY PROFESSOR L. EDINGER,

Frankfurt, a.M.

HAVING recently devoted much time to the study of the cerebellum from the standpoint of comparative anatomy I am glad of the present opportunity to present a preliminary account of my researches.

(1) QUANTITATIVE CONSIDERATIONS.

The degree to which the cerebellum is differently developed varies directly according to equilibratory needs as I have formerly suggested. I would now point out that, for instance, myxine which lives either quite parasitically in other fish or firmly attached by its sucker to stones, has in effect no cerebellum. Again, that in the land tortoises the cerebellum attains only half the size which it reaches in the active swimming water tortoises and further, what is particularly interesting, that the spotted flat fish which practically remain quietly in sand have but minimally developed cerebella compared to other species of fish to which they are most closely allied.

Finally, as I have previously proved, whereas lizards only have thin and slender lamellæ, their near congeners, the crocodiles, possess large doubly recurved cerebellar folds.

(2) CORTEX AND NUCLEI.

All animals possessing a cerebellum exhibit a perfectly typical cortex and all have lateral intrinsic nuclei, which probably correspond to the dentate nuclei of mammals since the anterior (superior) peduncle springs from their interior.

The median (fastigial) nuclei are clearly defined, first in birds and mammals, though they are already indicated in *Chelone midas*, the edible turtle, which is a powerful swimmer.

The hemispheres are first developed, when as in mammals, a connection with the cerebrum is established. In the bird this does not exist.

(3) Peduncles.

Since, thanks to so many investigations, we now know the course of the fibres in the peduncles I have attempted to make a completely new schematic arrangement of the same.

It is not correct to speak of the lower vertebrates as possessing the superior (anterior), middle and inferior (posterior) peduncles of the mammalia. In the former there is only one peduncle from which tracts branch off frontally and caudally.

The general schematic subdivision which I now propose is quite different to that generally given, and, moreover, has the advantage of applying both to the lower and higher vertebrates.

(*A*) Afferent Tracts.

The cerebellum is the receptive apparatus for tracts originating in the first end stations of various afferent nerves *tractus nucleo-cerebellaris*, as follows:—

(*a*) In the spinal cord, from Clarke's column by the dorsal and ventral spino-cerebellar tracts.

(*b*) From the posterior column nuclei by the anterior and posterior fibræ arcuatæ as I have already (in 1884) shown. It should be now added that both these tracts are present in all animals from selachians to man.

(*c*) From the first end stations of the bulbar afferent nerves, at least of the vagus, acusticus and trigeminus.

As regards the nervus acusticus the position is somewhat complicated by the fact that the nucleus vestibularis lies within the cerebellar region in consequence of which the tract passing from it to the cortex must be very short.

(*d*) From certain direct root fibres.

These latter I first observed anatomically in selachians, in which animals I was able to demonstrate, after simple division of the vagus, acusticus and trigeminus nerves, that strongly marked degenerated tracts entered the cerebellum.

(*e*) From the end stations of the optic nerves in fishes, birds, amphibians and reptiles.

Collectively the afferent paths in mammals run in the most caudal peduncle, and in its mesial portion, they cross to the contralateral side of the cerebellum either wholly or in part (*i.e.*, by far the large majority).

(*B*) Efferent Tracts.

The cerebellum sends out a powerful tract of fibres, which terminates in the tegmentum. I term it the *tractus cerebello-tegmentalis*. Large nerve cells of the motor type lie wherever its fibres reach.

The tractus cerebello-tegmentalis is very obvious in all vertebrates, and appears to emerge always from the intrinsic nuclei, but this can be finally established by the degeneration method only. It is also possible that beginning as fibres from the Purkinje cells its fibres pass through the intrinsic nuclei here and there.

In my opinion the following parts of it can be differentiated:—

(*a*) *Tractus cerebello-tegmentalis bulbi.*—The fibres composing this bundle end in the region of the lateral nucleus of the dorsal reticular region of the medulla oblongata. This has been definitely ascertained, especially in the case of birds and teleosteans, also in mammals (doubtfully in amphibians).

(*b*) Certain fibres reach as a tractus cerebello-tegmentalis spinalis into the upper part of the spinal cord. To this bundle belong the fibres described by Thomas, and possibly also the tractus vestibulo-spinalis, which passes downwards from Deiter's nucleus.

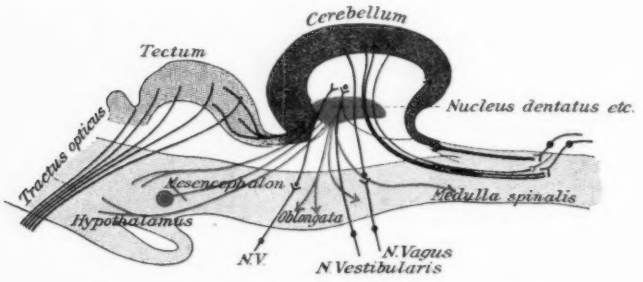
(*c*) *Tractus cerebello-tegmentalis mesencephali.* — This tract consists in selachians and teleosteans in a dorsal uncrossed, and a ventral crossed path ending in the teg-

mentum of the mid-brain. The latter portion of it is present in all vertebrates, from petromyzon to man. From the reptilia upwards the scattered ganglia of the tegmentum are differentiated as special nuclei, the nuclei rubra of the mesencephalon.

(*d*) *Tractus cerebello-tegmentalis diencephali.*—The fibres of this tract run with those just described and are always uncrossed. They end further frontally in the hypothalamus.

The tracts described under *c* and *d* constitute in mammals the superior cerebellar peduncle, the crossed portion of which ends in the red nucleus and the direct portion in the thalamus.

In teleosteans the thalamic portion is by far the



largest tract in the whole brain. Powerfully developed bundles pass without crossing the mid-line from the cerebellum into the hypothalamus, which, for their accommodation, is developed into two considerable swellings, the *lobi inferiores.*

The same condition exists also in selachians, but much less strongly marked. In petromyzon and amphibians with minimally developed cerebella, I have not found this thalamic expansion.

Thus, it will be seen that, without in any way contradicting the position which has been arrived at by our best authorities, I would substitute a wider and, at the same time, a simpler scheme of the cerebellar system.

ON THE MOVEMENTS OF THE TONGUE IN HEMIPLEGIA, AND FROM CORTICAL STIMULATION—AN APPARENT PARADOX; WITH A NOTE ON THE MOVEMENTS OF THE TONGUE AFTER PARALYSIS OF ONE HYPOGLOSSAL NERVE.

BY CHARLES E. BEEVOR, M.D., F.R.C.P.

In hemiplegia the tongue, when affected, is protruded towards the paralysed side, or, in other words, towards the side opposite to the lesion in the internal capsule or the cortex. On the other hand, electrical stimulation of the cortex cerebri in the monkey produces protrusion of the tongue to the side which is opposite to the cortex stimulated. Assuming that we may compare the brain of the monkey with that of man, we apparently have the same movement of protrusion of the tongue to the opposite side produced by both paralysis, and stimulation, of the cortex, which sounds an absurdity.

Taking first the case of hemiplegia, it has been known since the time of Abercrombie [1] that in hemiplegia when the patient was told to put out his tongue, it was protruded towards the paralysed side. This symptom has been seen so often, and has been so frequently confirmed, that there can be no doubt that in paralysis due to a lesion of the cortex or of the internal capsule—let us say—of the left hemisphere, the tongue when protruded goes over to the right side.

Taking next the question of the electrical stimulation of the cortex cerebri in the monkey, it was shown by Sir Victor Horsley and myself in a paper [3] read before the Royal Society twelve years ago, that stimulation of the cortex produced protrusion of the tongue to the opposite side, so

that with stimulation of the left cortex the tongue was protruded with the tip towards the right side. The movement was apparently the same movement as that which is produced when the left cortex in man is paralysed in hemiplegia. What, then, is the explanation of this apparent paradox?

I think that the explanation will be found in the investigation of the different movements which we obtained by stimulating different parts of the cortex in the monkey.

The area in the cortex of the monkey (*macacus sinicus*) from which movements of the tongue can be elicited by electrical stimulation, lies chiefly in the lower part of the ascending frontal convolution, extending from the horizontal level of the angle of the inferior præcentral sulcus above, down to the fissure of Sylvius. In the upper part of this area is represented the movement of protruding the tongue towards the opposite side. In the lower part of this area, stimulation of the anterior half in front of the sulcus frontalis transversus inferior gives the movement of advancing the tongue straight, while in the posterior half we obtain the movement of retraction of the tongue. A third movement, viz., rolling over of the tongue to the cheek on the same side as the cortex stimulated was observed on stimulation of the part between the upper and lower parts of the area, but with this we need not concern ourselves. The two movements which we have to take into account are the protrusion of the tongue straight, represented in the anterior half of the inferior part of the "tongue area," and the protrusion of the tongue to the opposite side, represented in the upper part of this area.

Now, these two movements are of a character which is quite distinct and different, and they are represented in different parts of the cortex. To show the different character of these two movements, and also to show what share was taken by the two halves of the tongue in producing them, the tongue was divided (under deep anæsthesia) into two halves along the raphé down to the hyoid bone, so as to form two independent organs. It was then seen that when

the area for protrusion of the tongue straight was stimulated in the left hemisphere, the two halves of the tongue were protruded straight, and each half of the tongue performed exactly the same evolution, and in no case could preponderance of action be shown to take place on either side. It follows, therefore, that these movements are truly bilaterally represented"[4], or in other words, that precisely the same movement could be obtained, when either the right or the left cortex was stimulated. When, however, the upper part of the area in the left hemisphere, where the protrusion of the tongue to the opposite side is represented, was stimulated, the following peculiar movements were seen. The half of the tongue, on the same side as the cortex stimulated, was seen to be advanced beyond the teeth, while the half of the tongue on the opposite side was seen to be retracted. The resultant of these two movements on an entire tongue would be the movement of protrusion with the tip to the opposite side. The opposite character of the movements of the two halves of the tongue was strikingly shown when an epileptic fit was produced, remarkable " see-saw " contractions being obtained, the half of the tongue on the same side being shot out of the mouth at the same time as the opposite half was retracted.

It has been the custom to assume that the left cortex governs the right half of the tongue, and the right cortex governs the left half of the tongue, exclusively. It has also been stated that the deviation of the tongue in hemiplegia towards the paralysed side is due to the want of action of the muscles of the paralysed side, the tip being directed by the muscles of the healthy side, which will not act in a straight line when not antagonised by their fellows. It should be remembered that, according to Dr. Hughlings Jackson, it is the movements and not the muscles which are represented in the cortex cerebri, and according to our experiments we would say that in every movement of the tongue produced by cortical excitation, both halves of the tongue always take part.

It has been stated above that in our experiments, when

protrusion of the tongue was produced by electrical excitation of the cortex of one side only, no preponderance of action could be shown to take place on either side, and from this it would follow that, if this area for the representation of the tongue on one side were destroyed, the same movement of the two halves of the tongue could be produced by stimulation of this area on the intact hemisphere. If this be so, it is difficult to understand why, with a lesion like hemiplegia, which affects only one hemisphere, the movement of protruding the tongue straight should ever be interfered with, as long as the other hemisphere is not affected.

The explanation as to why the tongue is ever deviated in the movement of protrusion straight is very difficult to give; but I would suggest that the way out of the difficulty may be found by examining the representation of other movements situated near the middle line of the body; and the two movements which I would take are the movements of elevation of the soft palate and the movement of flexing the spine by the recti abdominis. Taking first the movement of elevation of the soft palate, we showed in our experiments [5] that, with minimal electrical stimulation of the cortex of one hemisphere, elevation of the soft palate towards the opposite side could be obtained more frequently than bilateral elevation of the palate, *i.e.*, in the proportion of eleven times to four. Also a case has been recorded by Dr. Jackson and myself of paralysis of one half of the palate from a lesion of the internal capsule, and yet I believe that it is quite impossible for any one to imitate the movement by voluntarily drawing up the soft palate to one side. This, I think, shows that to draw up the palate straight in the normal way one hemisphere is not sufficient, but we must use both hemispheres to put equally into action the levator palati of each side.

With regard to the rectus abdominis, Sir Victor Horsley and Professor Schäfer [6] found on stimulating the cortex electrically, and Sir Victor Horsley and myself [2] found by exciting electrically the internal capsule in the monkey, that the rectus abdominis of the opposite side only was thrown into action; that is to say, that the cortex of one

side could only make the opposite rectus abdominis contract to produce the unilateral movement of abducting the trunk to the opposite side. It would therefore seem that for the bilateral movement of flexing the spine, as in rising up from the prone position, where both recti abdominis are in use, both hemispheres must take part.

I would therefore suggest that in the movement of protrusion of the tongue straight, both hemispheres take part; but the necessity for both to act is to a less degree than in the case of the elevators of the soft palate, and still less degree than in the case of the recti abdominis; and that, although both halves of the tongue are represented in each hemisphere, yet in voluntary efforts when one hemisphere is unable to act, the other acts more strongly on the opposite half of the tongue, and carries it over towards the paralysed side.

It will now, I think, be apparent that the explanation of the paradox is, that the movement of protrusion of the tongue to one side which occurs in hemiplegia is not the same movement as the movement of protrusion to one side produced by electrical stimulation of the cortex in the monkey. In the first case the patient is told to put his tongue out straight, and in the monkey this movement would be produced by exciting the anterior inferior part of the cortical area for the tongue; while, in the other case, the movement of protrusion of the tongue to one side is obtained in the monkey by exciting the upper part of the "cortical tongue area." In right hemiplegia the patient wishes the tongue to *come out straight* and it goes to the paralysed, *i.e.*, right, side without his knowing it, and in stimulation in the monkey we intend the tongue *to go to the right side* by stimulating the upper part of the left "tongue area," and it does go to the right.

Another point is that, if the deliberate movement of protruding the tongue, let us say, to the right be produced by the action of the left cortex, in right hemiplegia due to a lesion of the left cortex or of the internal capsule, there ought to be some paralysis of this movement. I have for some time made a point of testing this movement by asking

patients to put the tip of their tongue into the cheek of either side, and I have found that, in those cases where in right hemiplegia the tongue when protruded goes to the right, there is more difficulty in putting the tongue into the *right* cheek than into the left.

Movements of the Tongue when One Hypoglossal Nerve is Paralysed.

In connection with the question of the movements which were obtained when the tongue was bisected and the cortex was stimulated, I might record the movements which are possible when the left hypoglossal nerve is paralysed. When the patient was told to protrude the tongue *straight* the tip went a little to the left of the middle line to about the level of the left canine tooth, but when he was told to carry the tongue past this point to the left he was not able to do so, and he had no power to put the tip of the tongue into the left cheek. The reason of this is, that the left half of the tongue being paralysed the requisite muscles for retracting the left half of the tongue were absent (see page 488), and no power of the will could make the muscles of the right half do this. On being told to protrude the tongue to the right it was seen that though the tip of the tongue could be twisted round so as to touch the teeth of the right lower jaw as far back as the second molar, it was not possible to protrude the tongue beyond the teeth so as to touch the cheek. Protrusion of the tongue to the right is produced according to our stimulation experiments [3] by retraction of the right half and advancing of the left half. In this case the left hypoglossal was paralysed, the left half of the tongue could not be advanced, and all the movements of protrusion to the right had to be done by the right half of the tongue, whose action in this movement was that of retraction which could not possibly advance the tongue across the boundary of the teeth, and which the will had no power to alter.

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[2] Horsley and Beevor. *Phil. Trans. Roy. Soc.*, 1890, vol. 181, B.
[3] Horsley and Beevor. *Phil. Trans. Roy. Soc.*, 1894, vol. 185, B., p. 55 *et seq.*
[4] Horsley and Beevor. Same paper, p. 56.
[5] Horsley and Beevor. Same paper, p. 62.
[6] Horsley and Schäfer. *Phil. Trans. Roy. Soc.*, 1888, vol. 179.

TUMOUR OF THE CEREBELLUM IN A DOG, ASSOCIATED WITH FORCED MOVEMENTS.

BY FREDERICK E. BATTEN, M.D.

(From the Neurological Research Department, National Hospital, Queen's Square.)

(1) Introduction.
(2) History of Case and Observation of Clinical Symptoms.
(3) Pathological Appearances, Naked Eye.
(4) Microscopical Examination.
(5) Symptoms Observed in this Dog Compared with those seen after an Experimental Lesion.
(6) The Nomenclature and Description of Rotation as Applicable to Animal and Man.
(7) Other Pathological Observations on Animals, Presenting Cerebellar Symptoms.
(8) Conclusion.

(1) Introduction.

It is well known from experimental investigation that injury to or removal of portions of the cerebellum in lower animals gives rise to a definite train of symptoms, of which possibly the attitude assumed, the rotation of the animal on its long axis and the circus movements are the most striking, although not the most characteristic and constant features, of such a lesion.

Apart from experimental lesions, some observations have been made in animals, in which the symptoms during life pointed to a cerebellar lesion, and on investigation, a cerebellar lesion was found; such are the cases reported by Herringham and Andrewes, Risien Russell, and Thomas Lewis. Rotation on the long axis was not present in any of these cases, and circus movements were present only in the case reported by Thomas Lewis.

In all these reports, atrophy of *both* lateral lobes of the cerebellum was present.

In the first place it will be well to describe the symptoms which the dog presented and the pathological appearances, and afterward to compare the symptoms with those which have been observed after experimental lesion.

(2) History of the Case.

The following is the history of the dog, as obtained from Mr. Prime, the veterinary surgeon, under whose care the dog came. Mr. Prime recognised the unusual nature of the symptoms, and sent the dog to Mr. Hobday for a further opinion. The dog, a black mongrel terrier, aged 18 months to 2 years, was bought in London three weeks before for two shillings, and was being trained as a jumping dog—the trainer had not noticed the animal to have had any bad falls on the head. Five days before sending the dog to Mr. Hobday, Mr. Prime had noticed slight loss of control of the hind quarters, gradually increasing till three days after the first symptoms had appeared the dog could not stand. He carried his head to one side, and rolled over when excited.

The dog was seen with Mr. Hobday by Dr. Gordon Holmes and myself, and the following account of the symptoms is based on notes and drawings made by Dr. Holmes and myself at the time of, and immediately after, seeing the animal:—

In one or two points the accounts differ, more especially in regard to the attitude. It is of interest to note that although we are describing the same phenomenon, and we are in perfect agreement as to the direction of the rotation, Dr. Holmes says, "the animal at once rolled over to the left," whilst my description is "the dog at once rotates on its long axis from left to right;" both of us at once qualify the statement by a description of the movement, which leaves no doubt as to the agreement, but the fact demonstrates how misleading the terms "rotation to the right or left" may be, and that other nomenclature is needed to describe these movements. The dog was unable to stand. If held up by the scruff of the neck and by the skin over the rump, he squirmed about so that it was hardly possible to hold him.

If stood on his hind legs, with the fore limbs and head held between the knees, he could support his weight.

Attitude.—When left alone the dog lay on the ground on his right side, and with the right side of the face against the ground, the left side being turned up. The whole body was curved, the concavity being ventralwards, and on the left side of the body. (Plate I.)

The position of the head was very marked and constant. The head was flexed so that the right ear approximated to

PLATE I.

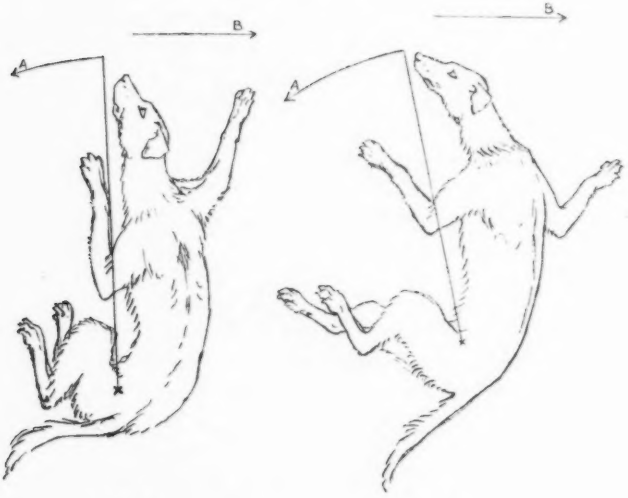

FIG. 1. FIG. 2.

PLATE I.—FIGS. 1 AND 2.

These figures indicate the position assumed by the dog as seen from above, the animal lying on the floor on its right side.

Fig. 1 represents the position as reconstructed from a rough sketch of mine made at the time. (F.E.B.)

Fig. 2 represents the position as reconstructed from a sketch made by Dr. Holmes.

The letter *A* indicates the circumference of the circle, and the direction of the "circus movements" which the dog performed.

The letter *B* indicates the direction in which the dog rotated on its long axis along the stable floor.

For the reconstruction of the drawings from the rough sketches I am indebted to my brother (Mr. J. D. Batten).

the right shoulder. The head was rotated on its long axis so that the left side of the face was turned up. The head was kept in close approximation to the ground. There was no great rigidity of the neck muscles. (Plate I., figs. 1 and 2.)

Limbs.—The right fore-limbs were abducted and placed on the ground, at an angle of about 45° to the long axis of the head. The left fore-limb was flexed, and the fore part lay parallel with the long axis of the dog. (See plate I., fig. 1.) Dr. Holmes and myself are not in absolute agreement on this point, and it may be well to give his description of the attitude in his own words : "The right fore-limb lay partly under, partly behind the body—the humerus seemed to separate the shoulders from the floor, while the arm and paw lay flat on the ground." (See plate I., fig. 2.) The hind legs were generally flexed, but at times became extended, the right being generally more extended than the left. The legs were not spastic, for they could be passively flexed with the greatest ease ; on the other hand, when handled, they were generally found to be rigid, this rigidity seeming to be due to volitional effort in the attempt to avoid forced movement. Dr. Holmes considered the right hind-limb was distinctly stiffer than the left.

Movements.—(1) Forced movement of rotation on long axis.—If the dog was undisturbed he would lie still in the position above described for some time ; but under emotional stimulus, or if the right fore-limb was brought in parallel to the long axis of the body, so that it no longer formed a support, the dog at once rotated on its long axis from left to right ; *i.e.*, regarded as a corkscrew or common screw,[1] he rotated as an outcoming screw, or, in other words, the right shoulder moved forwards across the trunk until the same relative position was reached. At times the rotatory movement would occur two or three times, so that the dog rolled over and over along the stable floor on his long axis, and only came to rest when the right fore-limb was sufficiently far out to prevent further rotation. The rotation was so rapidly performed that it was impossible to describe any movement of the body during its execution.

[1] The head of the animal corresponded to the head of the screw or the handle of the corkscrew.

Dr. Holmes, in his description, says the animal at once rolled over to his left (*i.e.*, when lying on its right side the limbs arose and passed across the trunk and then the trunk over the limbs).

We are in perfect agreement as to which way the dog did roll, and the difference in description depends upon the fact that one is accustomed to regard an animal in the prone position, a man in the erect or dorsal position.

I, myself, am of opinion that the method of describing rotation in the terms "the right (or left, as the case may be) shoulder moves forward" is that which is least liable to error.

The ingoing and outcoming screw is also a perfectly clear method of describing rotation to all who are familiar with the use of an ordinary screw and do not wish to quibble as to a male or female, and a right- or left-handed screw, the head of the animal representing the head of the screw.

(3) Circus Movements.

Apart from the rotatory movement on its long axis, the dog tended to move round in a circle on the floor anti-clockwise, *i.e.*, if a watch were placed on the floor with the face upward, the movements would be in a direction opposite to that of the hands (plate I, figs. 1 and 2), the direction of the circus movement is indicated by the circumference of a circle with an arrow mark. During these movements the dog, lying on his right side, kept the head on the ground and "paddled himself round with short movements of the forelimbs in a circle, whose centre corresponded approximately to the hip joints and with a radius extending from here to the tip of the nose." To what extent the movement was volitional or to what extent it was a forced movement was impossible to tell.

The dog appeared in good health, had a good coat, and was friendly and responsive. There was no paralysis of the face, no nystagmus, no ocular paralysis. The knee-jerks were present and equal.

The dog was killed with hydrocyanic acid and a *post-mortem* immediately made.

(4) Pathological Appearances.

On superficial examination the cerebral cortex, the surface of the cerebellum and spinal cord, appeared quite normal. On examination of the medulla and under surface of cerebellum, a tumour was seen to occupy the medulla on the right side and involved the region of the olive and inferior peduncles of the cerebellum on that side. The tumour had obliterated the external configuration of the structure in this neighbourhood, but did not affect the pyramid. On section, the tumour was seen to involve the central and peripheral portion of the right lateral lobe of the cerebellum together with the flocculus, restiform body, and the eighth nerve in its course and exit. The whole of the right side of the medulla was much larger than the left, and the tumour involved the region of the inferior cerebellar peduncles, the upper and outer portion of the olive, and the ascending root of the fifth. The total length of the tumour was 1·4 cm. On section, the tumour was firm and more vascular than the surrounding brain tissue.

Plate II. shows the position of the tumour as seen from the base of the brain. Figs. Nos. 1—8 show the position of the tumour as seen on section by naked-eye examination.

(5) Microscopical Examination.

The pons, cerebellum, and medulla were cut in series and stained by the Weigert-Pal, and van Geison method.

The extent and position of the growth.—A section of the cord at the level of the decussation of the pyramids showed no change.

In a section at the level of the posterior column nuclei, it was obvious that the right side of the cord was larger than the left, but on microscopical examination no obvious growth could be seen, nor was the cause of this enlargement of the cord on the right side apparent.

In a section at the lowest part of the fourth ventricle the growth appeared on the periphery of the cord growing into the substance of the cord from the vessels of the pia mater. In a section at the level of the inferior olive the growth involved a considerable portion of the right half of

PLATE II.

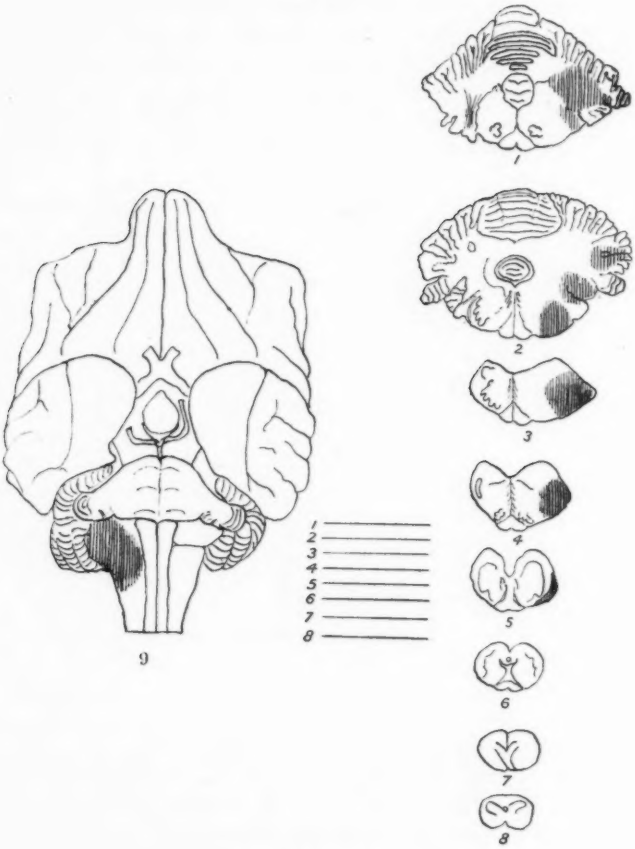

PLATE II.—FIGS. 1 TO 9.

1 Base of the brain of the dog. The shaded area indicates the position of the tumour, involving the medulla and under-surface of the cerebellum on the right side. The horizontal lines numbered 1—8 indicate the line of section, and the shaded areas in figures 1 to 8 show the position of the tumour as seen with the naked eye.

the medulla. It did not, at this level, invade the olive itself, but involved the region of the direct cerebellar tract and the substantia reticularis (plate III., fig. 6).

About this level the growth became much more extensive and involved the restiform body, the ascending root of the fifth and the outer part of the olive, but at no point was the pyramid involved (plate III., figs. 5 and 4). The eighth nerve on the right side was involved in the growth and the peripheral portion of the seventh nerve in its course through the medulla was also involved, the central portion remaining intact. The sixth nerve was not involved in the growth (plate III., fig. 3).

The region of Deiters' nucleus was not actually involved in the growth, but only a few multipolar cells of this nucleus could be seen, and these were more numerous on the right side than the left.

The growth extended up into the cerebellum, involving the cerebellar cortex in the region of the flocculus and the region of the dentate nucleus (figs. 2 and 1). The growth involved the fifth nerve on the right side, but did not destroy it, for it stains relatively well by the Weigert-Pal method.

In a similar manner the superior cerebellum peduncles were affected to a slight extent on the right side.

On the left side there was at this level a small mass of growth completely separated from the main growth. This growth involved the folia of the cerebellum, but did not extend into the middle of the cerebellum or involve the fifth nerve.

With regard to the cranial nuclei it is difficult to state that there was any definite change. The cells in the right hypoglossal nuclei were not so numerous as those in the left. The nuclei of the vagus were well stained. The sixth nucleus appeared normal, as did also many cells in the seventh, in spite of the fact that the intramedullary portion of the nerve passed through the growth.

Many fairly normal nerve cells could be seen lying within the growth.

Nature of the growth.—Sections were stained by van Gieson's method. Under the low power the growth is seen

PLATE III.

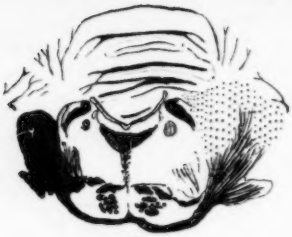

FIG. 1.

FIG. 4.

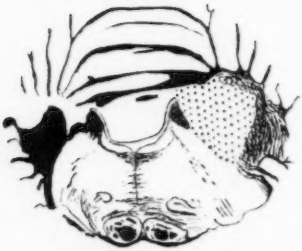

FIG. 2.

FIG. 5.

FIG. 3.

FIG. 6.

PLATE III.—FIGS. 1 TO 6.

FIG. 1.—Section through pons and cerebellum. The situation of the tumour is indicated in all the sections by the dotted areas.....

The tumour at this level occupies the central portion of the right lateral lobe of the cerebellum. The middle peduncle of the cerebellum, and the superior peduncle, to a limited extent, and ventrally involves the greater part of the fifth root, but does not completely destroy it.

FIG. 2.—Similar to the above, but below the fifth root.

FIG. 3.—Section through the medulla at its junction with the pons. The great mass of the growth is situated on the right side of the medulla and lower part of the cerebellum. It involves, at this region, the inferior peduncle of the cerebellum, the flocculus, the eighth nerve, and the seventh, in part of its course. The outer part of the right olive is also involved. A small mass of growth is also seen in the left lobe of the cerebellum, growing in from the pia mater, and involving the cortex of the cerebellum, but not extending into the middle of the lobe.

FIGS. 4, 5, and 6.—Sections through the medulla indicating the extent of the growth at various levels. The growth involves the direct cerebellar tract and restiform body, the ascending root of the fifth nerve, and the outer part of the olive, but does not involve the pyramidal fibres.

to be composed of small round cells, darkly stained, and larger oval cells lightly stained with a nucleus; these cells are fairly uniform in size.

The cells are massed for the most part round vessels, and hence the vessels are clearly picked out in their course through the growth. In portions of growth in which the vessels were cut in transverse section a vessel could be seen in the centre surrounded with layers of cells. No giant cells could be seen.

Towards the periphery of the growth, the extension was along the walls of the blood vessels.

The growth appeared to have started on the outer surface of the cord probably in the walls of the vessels of the pia mater and gradually to have invaded the medulla along the course of the blood vessels. The growth would probably be classified as a sarcoma growing from the outer sheaths of the blood vessels.

As another observer to whom I have submitted the specimen is not in agreement with the above statement, it may be well to state his views:—

Dr. Lazarus Barlow has kindly furnished the following report: "From the peculiar distribution around the blood vessels, from the character of the cells and particularly from the fact that I find a fair number of polymorphonuclear leucocytes intermixed with the chiefly lymphocytic elements forming the masses, I have come to the conclusion that the condition is inflammatory and not neoplastic. From the neoplastic point of view, the mass could only be angiosarcomatous, and I am convinced that it is not of that nature."

(6) Symptoms observed in this Dog Compared with those seen after Experimental Lesions.

In order to compare the symptoms seen in this dog with those observed after experimental ablation, the following table had been arranged in terms of ablation of the right lateral lobe of the cerebellum.

From a comparison of the above results it will be seen that the direction of rotation in this dog corresponded to the experimental results of Luciani, Ferrier and Turner,

TABLE I.

Ablation of the Right Lateral Lobe of the Cerebellum.

	Luciani.	Ferrier and Turner.	Russell.	Thomas.	Dog Tumour of Right Side.
Rotation	To right. Right shoulder moves forward. Outcoming corkscrew	To right. Right shoulder moves forward. Outcoming corkscrew	To left. Ingoing corkscrew	To right, *i.e.*, right shoulder moves forward. Outcoming corkscrew.	To right, *i.e.*, right shoulder moves forward. Outcoming corkscrew.
Reeling	To right	.. ..	To left	.. ..	Circus movements to left, *i.e.*, anti-clockwise.
Attitude	.. ..	Head inclined to right side and rotated to the left	Right side of face turned up. Right ear approximate to right shoulder	Head to right side shoulder. Left side of face turned up	Right ear approximated to right shoulder. Left side of face turned up.
Concavity of Spine	To left	None	To right	To the right, lies on right side	To the right, lies on right side.
Ataxia	On right side	General	Right side	Right side	General.
Weakness	On right side	None	Right side and posterior limb of opposite side	Right side	No true weakness, inability to stand due to forced movements.
Tone	Loss of tone on right side	No loss	Rigidity of right side ..	General rigidity, over action of right side	General slight rigidity.
Anæsthesia ..	None	None	Same as motor paresis		
Ocular paralysis ..	Skew deviation : Right, in and downwards. Left, up and out	Skew deviation : Right, in and down. Left, up and out	Left eyeball downwards and outwards	Deviation to the left	None.
Nystagmus ..	.. ..	.. ..	To the right	.. ..	None.

and Thomas, but were in the opposite direction to those described by Russell.

With regard to the attitude assumed the position in the dog corresponded to the experimental results of the same observers and differed from Russell in the rotation of the neck.

The direction of the concavity of the spine corresponded to that observed by Russell and Thomas, and was the reverse to that seen by Luciani.

The other symptoms—the ataxia, weakness, rigidity of muscles—were general in the dog and not localised as in most of the experimental results.

No ocular paralysis or nystagmus was observed in this dog.

Seeing, however, that the tumour involved the eighth nerve on the right side in the dog, it is interesting to compare the result with those obtained by Russell on section of the nerve on the right side.

In this animal after section of the eighth nerve, the dog rotated to the right (outcoming corkscrew), the reverse direction to that obtained by Russell on ablation of the right lobe. The dog fell to the right side, had concavity of the spine to the right, and performed circus movements to the right. There was no definite ataxia, weakness, loss of tone, or anæsthesia, nor was any definite position of the head assumed.

Section and irritation of the nerve gave the same result.

Dr. L. J. Muskens gives a good account of circus movements and rotation as seen both in the lower and higher vertebrates and his observations correspond with the movements observed in this dog. The ocular phenomena to which he pays special attention were not present in this dog.

(7) The Description of Position as Applicable to Man and Animals.

I have already made some allusion to this subject and the difficulty which arises in describing rotation movement to right or left.

Rotation in the long axis should be described in terms of the right or left shoulder moving forward, or in terms of an ingoing or outcoming screw.

Circus movements should be described in terms of the hands of a watch placed on the floor so that the observer can see the hands, clockwise or anti-clockwise.

(8) Observation on Animals Presenting Cerebellar Symptoms.

A few observations on pathological lesion of the cerebellum have been made in animals. The family of half-breed Persian cats with cerebellar symptoms investigated by Dr. Herringham and Dr. Andrewes are of great interest. These animals showed atrophy of the whole of the cerebellum.

Another interesting observation is that of Dr. Risien Russell on a puppy, one of a litter, all of whom were similarly affected. In this case the cerebellum was reduced to almost three-fourths its normal size.

A third observation is that of Dr. Thomas Lewis in a dog who had exhibited "circus movements" for at least four years, and in which there was a general diminution in the size of the cerebellum with shrinking of the dorsal surface of the lateral lobes.

One other case is of interest in the connection. A black pug, 1 year old, had never been very strong and had had distemper three months previously. Discharge from the left ear had subsequently been noticed; one month previously the pug had become unsteady on its legs and had been much worse for the last week—it had vomited and seems to have lost his vision.

When placed on the floor the dog started off to walk and walked round and round in a circle to the right (clockwise). The dog on this occasion could not be got to go in the opposite direction. The dog would wander round and round the room, always to the right, and was only stopped by coming in contact with an object which prevented his turning to the right. He would then stand in a dazed

condition, and as soon as the obstruction was removed on he would go on his walk. Both hind legs were very weak, and it needed but a touch to upset his balance.

A few days later he walked round in a circle to the left (anti-clockwise). This movement was not so marked as had been the circus movement to the right. The dog was killed, and on macroscopic examination of the brain and spinal cord nothing abnormal was seen. On microscopical examination changes were present in the cerebrum, the cerebellum and spinal cord, similar to those found in diphtheritic paralysis, *i.e.*, degeneration of the myelin sheath, and some alteration in the nerve cells when stained by the Marchi and Nissl methods.

(9) Conclusion.

Forced movements of the character seen in this dog and after experimental lesion are but rarely seen in man.

The symptoms presented by this dog were such as to lead one to believe that the lesion was situated on the right lateral lobe of the cerebellum and in this situation and in the medulla the larger lesion was found; a small tumour was, however, present in the left lateral lobe.

My best thanks are due to my friend, Dr. Gordon Holmes, not only for superintending the preparation of the section, but also for his help both in the clinical and pathological investigation of this case.

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[3] Thomas Lewis. *Brain*, 1904, p. 84.
[4] Luciani. *Il Cervelletto*, 1892.
[5] Muskens. *Journal of Physiology*, 1904, vol. xxxi., p. 204.
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[7] Russell. *Brain*, 1895, vol. xviii., p. 523.

THE CEREBELLAR ATTITUDE AND THE DENTATE NUCLEI.

BY E. FARQUHAR BUZZARD, M.D., F.R.C.P.

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A SHORT record of the following case, although not as complete as I should have liked, cannot fail to be of some interest in its bearing upon the causation of Dr. Hughlings Jackson's "Cerebellar Attitude." On February 22, 1906, I was asked to see a woman, aged about 60 years, with Dr. Bond and Mr. Donald Armour. The history was briefly as follows: In April, 1905, she had an attack of intestinal obstruction, caused by a malignant growth in the left iliac fossa. Mr. Armour explored the abdomen, and found the growth involved the sigmoid end of the colon, as well as many of the mesenteric and other glands in the neighbourhood. A colotomy was performed, and gradual convalescence supervened. A week after the operation a tertiary syphilide appeared on the calves of both legs, and a history of syphilitic infection was obtained from the patient.

In February, 1906, owing to the good condition of the patient and the diminished size of the tumour, the question of closing the colotomy wound was discussed, but a sudden change in the patient's health prevented this being carried out.

For a few days before I saw her, the patient complained of general malaise and appeared depressed, and on February 21 she became drowsy and stupid. On the morning of February 22 she was in bed in a completely unconscious condition. She lay flat on her back, with her legs rigidly extended and her arms rigidly flexed across her chest, the partially-closed hands pointing upwards towards her chin.

The neck was stiffly extended, so that in placing a hand behind the occiput and pressing forwards there was very little flexion of the head upon the trunk, but a tendency to lift the shoulders as well as the head. The abdominal muscles were not rigid. The pupils were moderately small, but reacted to light; the corneal reflexes were considerably diminished, the axes of the eyeballs were parallel in the midline, and the fundi oculorum were normal in appearance. Respiration was slow and rather shallow, chiefly thoracic in type, with equal movement on the two sides. The pulse was small, moderately rapid, and of low tension. The heart sounds were feeble, but no bruit was audible.

The abdominal reflexes were absent; the plantar reflexes were sometimes of the extensor type, but not well marked nor easily obtained. The tendon reflexes were brisk, but rather difficult to elicit, on account of the rigidity of the limbs.

Stimulation of the skin, by means of a pin, produced practically no response in any region.

The marked "cerebellar" attitude of the patient was taken into account in making the diagnosis of ponto-cerebellar thrombosis.

The patient became rapidly worse and died the following night, in spite of stimulative treatment and careful nursing.

At the autopsy, on the following day, the brain was removed and placed in 5 per cent. formalin. It showed no naked-eye evidence of disease, and the condition of the vessels was remarkably good, both at the base and over the cortex.

The abdominal tumour was found to be about the size of a large fist, and sections showed that it was a columnar-celled carcinoma, with a considerable amount of fibrous tissue.

After hardening for a fortnight in formalin, the brain was again examined and cut.

The forebrain appeared perfectly natural and healthy throughout. The brain, stem, and cerebellum, were cut in thin slices across their long axes; and, to my surprise, I found no evidence of disease in any part of the pons or

mesencephalon. The medulla, too, was quite firm and healthy-looking.

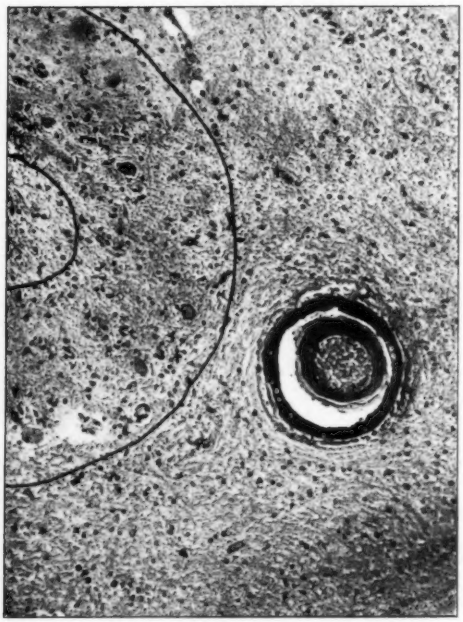
Both cerebellar lobes were natural as far as the cortex was concerned, but the dentate nucleus on each side was the seat of marked softening, evidently recent in origin.

This softening was limited to the immediate neighbourhood of the nuclei, and did not extend into the peduncles of the cerebellum.

An examination of the larger vessels in the neighbourhood revealed no naked-eye evidence of arterial disease. Sections of the dentate nucleus, with its surroundings, were cut from both sides and stained by van Gieson's and logwood-eosin methods. Portions of the pons and medulla were cut and stained by Busch's method for recent degeneration, but with no positive result.

The sections from the dentate nucleus region, when examined under a microscope, confirmed the fact that the thrombosis was limited to the nucleus and its immediate environment, and did not extend in the direction either of the peduncles or of the cortex cerebelli. They also supported the view that the thrombosis was of recent origin, inasmuch as there was no complete destruction of tissue, although both the nuclear cells and the surrounding elements were somewhat deficient in staining properties and indefinite in outline, compared to the cells in healthier parts around.

The cause of the thrombosis was very obvious under the microscope. All the arterial branches supplying the dentate nuclei showed disease of varying degrees of intensity and age. In many of them there was extensive thickening of the intima; but the most striking change was in the media, which was almost universally transformed into a calcareous ring, often brittle and not infrequently broken away from the inner coat. This calcareous substance stained a deep black, with the logwood stains and granules of the same material similarly coloured, were found marking out the course of the very smallest arterioles. Other vessels, with less intense changes in the media, sometimes showed a round-celled infiltration of the adventitia, suggesting that the latter was the



Photograph showing a diseased vessel with calcareous media and thickened interior; also one "tooth" of the dentate nucleus, with a number of cells, some of which are healthy.

seat of the earliest change in the morbid process. *Ante-mortem* thrombosis was evident in the lumina of some of the vessels, especially those in which the intima had undergone much proliferation.

Corpora amylacea were found in abundance along the course of the larger diseased arterioles.

Some sections were stained by the Nissl method with a view to studying the cell change more accurately. The cells of the dentate nucleus are normally round or slightly oval, the nuclei are well marked either in the centre or slightly to one side, and tigroid bodies are found in the surrounding protoplasm. More or less pigment is also frequently present. In this case practically no normal cell was found in the dentate nuclei, although in the same sections, at a little distance, perfectly healthy Purkinje cells were to be seen lining the granular layers of the folia.

The majority were somewhat shrunken, and frequently angular, and presented no definite nuclei or Nissl bodies, the protoplasm being faintly stained and apparently homogeneous or slightly granular (see Plate).

Remarks.

It is fair to assume that the only gross lesion in this case was the thrombosis affecting, and probably destroying the function of, the dentate nuclei. No macroscopical evidence, either of arterial disease or of softening, could be found anywhere else in the brain, and the labour of seeking microscopically for such lesions, with nothing to guide one as to locality, would have been very considerable and in all probability not justified by success. On the other hand, it is not reasonable to suppose that the lesion in the cerebellum was the cause of death. Some interpretation of what happened in the last few days of the patient's life is therefore necessary, and the one which is offered here appears to me to be, at any rate, not unwarrantable.

The fatal termination of cases of malignant growth of any part of the body is frequently similar to that which occurred in my case, as far as the development of a general malaise rapidly followed by a lapse into unconsciousness and coma is concerned. We are in the habit of attributing this

sequence of events to the excessive production of toxins by the neoplasm.

The toxæmia of this origin is associated with a lowering of blood pressure, which, we may fairly conclude, might easily establish a thrombosis in vessels whose walls were already in an advanced state of calcareous degeneration (and therefore inelastic), and whose lumina were encroached upon by a thickened intima.

In the case we are considering this appears to have occurred in the vessels supplying the dentate nuclei, a supposition which is supported by the fact that the thrombosis was recent, although the arterial disease was old. The thrombosis, indeed, was so recent that it had not produced degeneration (capable of demonstration by the Busch method) of nerve fibres arising from the cells of the dentate nuclei, although it was old enough to produce changes in the cells themselves and softening of the supporting tissue.

Assuming these considerations to be correct, the patient died a not unusual death from toxæmia, and the manner of death only differed from that of many other patients in the presence of a remarkable attitude assumed by the body during the last few hours of life.

In cases which die of toxæmia in the usual way, no gross lesions are found in the brain, so that it is only natural to conclude that the lesion of the dentate nucleus and the attitude of the body were in some way associated.

The next question for consideration is that of the relation between the lesion and the attitude. The *lesion* must be regarded as a negative one, *i.e.*, one which produced a loss of function of the part it affected; it does not appear to me conceivable that a thrombosis could produce an irritative lesion capable of maintaining an unaltered and unvarying position during more than thirty-six hours.

On the other hand, the *attitude* must be looked upon as a positive or active one, in that it was the result of increased tone in certain muscles of the trunk and limbs. It may be inferred, therefore, that the lesion destroyed an influence which normally opposed or inhibited, or modified, some other influence originating in another part of the nervous system, with the result that the latter influence was allowed free

play. Having gone so far, it is inevitable that we should consider how the facts in this case bear upon the cerebellar attitude of Hughlings Jackson and his views upon its production. According to Jackson's hypothesis, the cerebellar attitude is the result of the cerebral influx when it is allowed free play, and unopposed by the influx of the cerebellar subsystem. This view was founded chiefly on observations on cases of tumour of the middle lobe of the cerebellum, in which the cerebellar attitude was a remarkable feature of the clinical picture. Tumours are notoriously unsatisfactory as destructive lesions, and the cases were further complicated by the resulting hydrocephalus. In my case the lesion was almost as clear cut and defined as an experimental one, with the additional advantage that the cerebellar cortex was not injured in its production. On the other hand, the effect of the toxic agents upon the higher nerve centres must be taken into consideration. In this connection the condition of patients who have become unconscious, owing to the toxæmia associated with new growths, must be recalled. They do not assume or maintain any particular attitude, nor do their muscles present any evidence of increased tone, facts which argue in favour of the supposition that the incidence of the intoxication is chiefly, at any rate, on the highest level centres (pre-frontal cortex), and not to any large extent upon the middle level centres (Rolandic centres). If we allow this, and if we use the hierarchy of centres suggested by Jackson for the purpose of description, it is probable that in the cases of intoxication to which we refer, the middle level centres are still able to exert their influence upon the lower level centres; in other terms, the Rolandic cortex, on the one hand, and the cerebellar cortex on the other, are still able to influence the centres of the pons, medulla, and cord. In my case, the lesion of the dentate nuclei undoubtedly interfered with, at any rate, a considerable part of the cerebellar influence, and the resulting attitude may well be regarded as due to the more or less completely unopposed action of the cerebral middle level centres upon the lower centres, supposing that this view of opposing action on the part of the cerebral and cerebellar centres upon the lower level centres is a correct one.

ON THE RELATION BETWEEN LOSS OF FUNCTION AND STRUCTURAL CHANGE IN FOCAL LESIONS OF THE CENTRAL NERVOUS SYSTEM, WITH SPECIAL REFERENCE TO SECONDARY DEGENERATION.

BY GORDON HOLMES, M.D.

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THE practical significance of the amount of destruction of tissue that is produced by focal lesions of the central nervous system is often lost sight of, yet its importance is great, as it is very doubtful if regeneration of degenerated fibres of the central nervous system can occur. Apart from the fact that there are no satisfactorily substantiated observations that central fibres do regenerate, there is strong evidence that if the anatomical integrity of a central neurone is damaged its cell atrophies and disappears. Van Gehuchten [4], von Monakow [8], and Marinesco [6], have shown that, as a rule, when wholly central fibres are interrupted their cells of origin do not, like those of the peripheral neurones, pass from the stage of reactionary chromatolysis into that of reparation, but slowly disappear, so that the destruction of the neurone is irreparable and permanent.

It is, therefore, of considerable importance to ascertain, as far as possible, the amount of destruction of tissue that certain forms of disease or certain clinical symptoms indicate, for if the regeneration of the tracts of the central nervous system is not possible the recovery of function lost owing to the destruction of cells or fibres can result only from compensation by undamaged parts. It is obviously difficult to compare the intensity of clinical symptoms with the amount of structural change demonstrable under the

microscope, as entirely different standards are employed in estimating them. Yet pathologists must be frequently surprised at the little evidence of degeneration they find in regions or tracts to the affection of which considerable clinical symptoms were referable, and at occasionally meeting with evidence of severe disease when the symptoms that resulted therefrom were but slight or insignificant.

In pathological investigation the intensity of a focal lesion in the central nervous system is generally estimated by the amount of secondary degeneration found in the fibres of the tracts involved in the lesion, for as these constitute the functional tissue their condition alone need be considered in attempting to correlate structure and function; and the truth of the Wallerian law, that portions of fibres separated from their trophic centres degenerate, is incontestable. Whether the destruction of cells or the interruption of fibres is complete or not, is therefore determined by the presence or absence of secondary degeneration in the portions of the fibres separated from their trophic cells by the lesion, or in those which sprung from the gray matter involved in it.

It is certain, however, that the general application of this argument is not valid. Hitherto, in determining whether fibres are degenerated or not, we have relied largely on methods which demonstrate the condition of their myeline sheaths only, and have taken little count of the essentially functional part of the nerve fibres, the axis-cylinders and their component neuro-fibrils. It is conceivable that the myeline sheaths of fibres may degenerate, while the axis-cylinders remain relatively intact; this actually occurs in the focal lesions of disseminated sclerosis. On the other hand, it seems certain that as a rule when the myeline sheath can be shown to be normal by the methods of Weigert and Marchi, it may be assumed that there is no structural break in the fibre.

In investigating pathological material with this object in view the applicability of the methods of Marchi and Weigert must be considered. The former depends on the impregnation of certain products of degenerating myeline sheaths by osmic acid, and consequently can be appro-

priately employed only during that period after the onset of the degeneration, when these products are fully developed and before they are removed. Taking a safe margin it may be said that secondary degeneration in the central nervous system can be demonstrated by Marchi's method between two weeks and four months after the lesion. Weigert's chrome-hæmatoxylin stain reveals only the absence of myelinated fibres in the degenerated tracts, and is consequently the counterpart of Marchi's method. But as myeline may stain in the earlier stages of its degeneration, this method is applicable only from a certain period, about four weeks, after the commencement of the degeneration. Thus one or other of these methods can be employed for the demonstration of secondary degenerations from a short time after their commencement, but both reveal only changes in the myeline sheaths of the fibres.

The following cases have been selected to illustrate the occurrence of considerable loss of function owing to focal lesions in the central nervous system, without a corresponding degree of structural change, as indicated by secondary degeneration, in the affected fibre systems. For the use of the abstracts of the clinical notes, I am indebted to the kindness of the physicians to the National Hospital, under whose care the patients had been.

Case 1.—C. B., aged 42 years, was admitted to the National Hospital, under the care of Dr. Buzzard, in March, 1904. He had been healthy till the previous year; since then he had passed blood-stained urine, and was treated in a general hospital for tubercular disease of the bladder and kidneys. Nervous symptoms appeared about three months before admission, with slowly increasing weakness and sensory loss in his legs, and a girdle sensation around the upper part of his abdomen. He had been unable to walk for two and a half months. On examination, both lower limbs were found very rigid; there was great impairment in the range and power of their volitional movements, and they were frequently involved in severe reflex spasms. There was complete loss of tactile sensation to cotton-wool on the lower extremities, and considerable analgesia of the same distribution. The sense of position in the lower limbs was also diminished, the knee-jerks were exaggerated, and ankle clonus and extensor

plantar responses were obtained. While in hospital the spasticity and paresis of the lower extremities increased, the abdominal muscles became weak and rigid, and the sensory loss more marked. The tactile loss was complete to the cotton-wool test as high as the nipples, and a pin prick could not be distinguished below this level. A sharp prominence of the fourth and fifth dorsal spines developed, but there was very little rigidity of the vertebral column, and no tenderness on pressure. After leaving hospital he gradually became weaker, and lost all sensation and all power of movement below the level of the nipples. He died six months later, about a year after the onset of the paraplegia.

Autopsy.—The bodies of the fourth and fifth dorsal vertebræ were softened, and had collapsed. A large mass of caseous material projected into the vertebral canal and compressed the fifth dorsal segment of the cord, which was constricted and slightly discoloured. Examination of the compressed segment revealed some vascular congestion and neuroglial sclerosis, in addition to considerable destruction of the myeline sheaths; but no tract degeneration, either ascending or descending, was found in sections stained by Weigert's or Marchi's methods.

Case 2.—W. H., aged 48 years, entered the National Hospital, under the care of Dr. Farquhar Buzzard, in June, 1905. He had had a spinal curvature for fifteen years before he came under observation, but it was only five months before his death that his legs began to become weak. When admitted to hospital both lower limbs were spastic and weak; many of their movements were quite lost, and the lower half of the abdominal muscles were paralysed. He had been unable to stand for about a month. There was considerable, but not complete, anæsthesia and analgesia on the lower extremities and on the trunk to about two inches above the umbilicus. The tendon reflexes in the lower limbs were exaggerated, ankle clonus was obtained, and the plantar responses were of the extensor type. There was a sharp backward curvature of the vertebral column, its apex at the eighth dorsal spine. The evidences of spinal compression increased, and he died about nine weeks after admission from cystitis.

Autopsy.—Sharp kyphosis, its apex formed by the eighth and ninth dorsal spines. The eighth dorsal vertebra was very much diseased, and a large mass of tubercular caseous material projected from it into the vertebral canal and compressed the cord. The ninth dorsal segment, which was the most affected, was

narrowed and slightly discoloured and when examined microscopically its vessels were found congested, the white columns vacuolated, and there was some neuroglial sclerosis and increase of neuroglial nuclei. No trace of ascending or descending degeneration was revealed by either Marchi's or Weigert's method, though by the latter the compressed segment was palely and poorly stained, and numerous dots stained black by osmic acid were scattered through the whole cross-section of the segment in sections treated by the former.

Case 3.—F. T., aged 62 years, came under ths care of Dr. Ferrier in the National Hospital in May, 1905. Her legs had been weak for about a year, and for the past six months she had complained of severe reflex spasms in them; for at least a month she had almost complete loss of power and of sensation in these limbs, and incontinence of urine and fæces. On examination, the lower limbs were found very weak and slightly rigid, and the lower portions of the abdominal muscles did not contract in the attempt to sit up. There was considerable, but nowhere complete, loss of all forms of sensation on the lower limbs, and on the lower part of the trunk to midway between the umbilicus and xiphoid. Sphincter control was lost. The deep reflexes of the legs were brisk, the abdominal reflexes absent and the plantar of the extensor type. The fifth dorsal spine was prominent and tender to pressure. She died about three weeks after admission, the symptoms having progressed rapidly.

Autopsy.—Extensive tubercular disease was found in the bodies of the fifth and sixth dorsal vertebræ, and a mass of caseous material compressed the cord at this level. The seventh dorsal segment was the most affected; it was somewhat softened and discoloured, and, on microscopical examination, the vessels within it were found congested, and there was some perivascular small round-cell infiltration and slight glial sclerosis. The myeline sheaths of the compressed segment were much disorganised and stained feebly by the Weigert-Pal method, but there was no sign of ascending or descending secondary degeneration. By Marchi's method a few black masses, rather large and tending to accumulate round the vessels, were found in the compressed segments, but there was no secondary system degeneration above or below this level.

Case 4.—N. L., aged 15 years, was admitted under the care of Sir William Gowers in October, 1905. She then presented the typical symptoms of a pontine tumour. Her right limbs had

been weak for about a month, and, on examination, the movement of the right wrist and fingers were found almost completely paralysed. The paralysis increased rapidly; in November there was complete flaccid palsy of the right arm, and a month later of both right limbs, and in addition great weakness of the left limbs. The affection of the left limbs increased, and for at least a month before her death, in February, 1906, all four limbs were completely paralysed.

Autopsy.—A large infiltrating glioma of the pons was found, more extensive on the left than on the right side. It extended from the caudal end of the pons to the caudal limit of the crura cerebri. But despite the intensity and the long duration of the motor paralysis, no degeneration was found in the pyramidal or other tracts of the cord by either Weigert's or Marchi's method.

Thus in three cases of compression paraplegia, due to spinal caries, in which there was great loss of the power of movement and of sensation for a considerable time before death, no secondary degeneration could be found to indicate destruction by the lesions of even some of the fibres concerned in the normal activity of the functions which were lost. And the same statement, as well as the following arguments, holds equally well for the case of pontine tumour (Case 4).

A few similar cases are recorded in the literature. In the case published by Gauckler and Roussy [3] there was paraplegia with sphincter trouble, but without sensory loss, for two months before death. The only disease found was a sclerotic patch in the fourth dorsal segment of the cord, in which the greater number of the axis-cylinders persisted though the myeline sheaths had disappeared; no secondary degeneration resulted from it. (It seems possible that the lesion was a single focus of disseminated sclerosis.) Schlagenhaufer [9] records a case of complete paralysis of all limbs and complete sensory loss below the neck, due to compression of the upper cervical segments of the cord by an endothelioma; no secondary degeneration was found in the cord. In Marinesco's case [7] there was almost complete paralysis of all limbs for about two months before death, owing to compression of the cord by caries of the

atlas and axis and tubercular pachymeningitis; but no secondary degeneration was demonstrable in the cord by either the method of Weigert or of Marchi.

When, however, in the cases which I describe the nervous system at the level of the disease was stained by Weigert's method, it was evident that at least the myeline sheaths of many of the fibres of that region were considerably altered, or had even disappeared. They were found, especially in sections in which the fibres were cut longitudinally, swollen, irregular in calibre, and fragmenting. Further, in the cases of relatively short duration (Cases 2 and 3), the use of Marchi's osmic mixture revealed at the level of the disease the presence of fatty degeneration products, which had most probably arisen from the disintegrating myeline. But these methods give no indication of the state of the essential constituent of the fibres, the axis-cylinders. Even by ordinary methods of staining, however, it could be seen that these were much better preserved at the level of the active disease than the myeline sheaths, though all were not normal; a small proportion of them were swollen and irregular in calibre, but there was no apparent reduction in their number. To examine them more thoroughly the elective impregnation of Bielschowsky was employed, but, unhappily, only in Case 3 of the compression paraplegias reported above, and in the case of glioma of the pons. The results obtained were, however, controlled by the similar examination of the portion of a spinal cord which had been damaged by compression by an intrathecal tumour at the level of the first and second cervical segments. This case was very similar to the first three cases reported above, as, though there was complete loss of power in all limbs for at least a month before death, only a comparatively insignificant amount of degeneration was demonstrable in the cord below the level of the lesion by Marchi's method, and the segments stained by Weigert's method appeared normal.

In sections cut in the length of the fibres at the level of the maximum compression and stained by Bielshowsky's method, the remarkable preservation of the axis-cylinders

was at once visible. Apparently normal fibrils could be seen running through areas in which there was great tissue change, that is, vascular congestion, vacuolation of the tissue, and neuroglial sclerosis. So numerous were these axis-cylinders in even the thinnest sections, that there could be no suggestion of any numerical diminution, and only very rarely were any fragments seen that were not due to the oblique direction of the section. In both cases (Cases 3 and 4), however, a small proportion were swollen or irregular in calibre, but it seems probable that though diseased, the disease was not intense enough to interrupt their continuity.

The question at once arises, Is there any structural change in any part of these nerve fibres at the level of the compression, such as atrophy or degeneration of their myeline sheaths? It is difficult to give a direct answer to this question from Bielschowsky preparations alone, but it seems very probable that it was so. The difficulty consists in obtaining myeline sheaths satisfactorily stained, either in preparations in which the axis-cylinders are also visible, or in adjacent sections, as only sections which have been cut on a freezing microtome from unimbedded and unmordanted material can be used for Bielschowsky's method. The solution of the question was attempted by the aid of Weigert's and Fayersztajn's stains, and by that modification of Bielschowsky's method [1], by which this author asserts that the same results are obtained as by Fayersztajn's stain, that is that only axis-cylinders surrounded by myeline sheaths are coloured. These latter two methods were employed in sections intermediate between those in which the axis-cylinders were impregnated. The accuracy of this modification of his silver method recommended by Bielschowsky seems, in my experience, questionable; and though in normal tissues Fayersztajn's stain colours only medullated axis-cylinders, it seems probable that the sheaths of those which are disintegrating, or have recently disappeared, stain equally well. However, such evidence as these methods afford, and the more definite proof afforded by Weigert's stain, support very strongly the conclusion that the myeline sheaths of

many axis-cylinders which appeared normal had either disappeared or were undergoing disintegration at the site of the greatest intensity of the tissue change produced by the disease.

Bielschowsky [2] has observed a similar condition. In two cases of cerebral gliomata and in a region of spinal compression, due to tubercular vertebral disease, he found naked axis-cylinders. He assumes, however, that some of these represent regenerating fibres.

Little is known of the actual properties of the myeline sheaths of nerve fibres, but that they have some influence on the conduction of nerve impulses, the function of myelinated fibres, seems very probable. In disseminated sclerosis, in which the essential and practically the only structural affection of the nervous tissues consists in the disappearance of the myeline sheaths, without necessarily producing secondary degeneration the loss of function may reach extreme degrees. From this fact alone it may be assumed that the presence of myeline sheaths is necessary for the functions of the tract fibres of the cord as conducting strands, and that when they are lost there may be a physiological block in the fibres. This condition, from the point of view of function, is equivalent to a structural break in the fibres, but differs in the fact that the anatomical integrity of the axis-cylinder and its trophic cell remain unaffected, and that reparative processes would be consequently possible. That such return of function may occur has been frequently observed in the rapid recovery of power and sensation that occasionally follow the removal of tumours and the draining of tubercular abscesses which have produced symptoms of compression paraplegia.

The loss of function observed in cases of focal lesions, when not associated with secondary degeneration in the fibre systems which subserve the functions that are lost, may therefore be ascribed to the fact that the anatomical affection of these fibres at the site of the disease is incomplete, or limited to their myeline sheaths. On the other hand, it is conceivable that the local effect of the disease may be such that it can interrupt the conduction of physiological

currents, and yet is not sufficiently intense to damage the structure of any part of the fibre. The former view is, however, the better supported by pathological investigation, and seems the more reasonable; it is in accordance with the axiom stated by Dr. Hughlings Jackson thirty-six years ago, that "functional change must not be confounded with pathological change; although, of course, the two necessarily coexist," and that "structure, function and nutrition never exist separately." [5].

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ECTOPIA PUPILLÆ IN CERTAIN MESENCEPHALIC LESIONS.

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By ectopia pupillæ, or corectopia, is meant that condition of the eye in which the pupil is not in the centre of the iris. In the normal eye, it is true, the centre of the pupil and the centre of the iris are not always identical; the pupil is frequently deviated slightly downward and slightly inward, more rarely directly inward, and occasionally inward and upward. This deviation, moreover, may be unilateral or bilateral, and in the latter case it may be symmetrical or asymmetrical. To draw a hard and fast line between the physiological and the pathological is, of course, rather precarious, and one must be content to admit that some cases of ectopia pupillæ may have no pathological significance whatever.

Under certain well-recognised conditions, nevertheless, a more or less pronounced ectopic position of the pupil may be observed.

Thus, unilateral corectopia may occur, with conservation of the pupillary reflexes and a normal iris, except, possibly, for slight atrophic changes; apart from the frequent irregularity of the pupil no other anomaly is discoverable. Or it may be associated with an inflammatory condition, in which case the reactions of the pupils to light and other stimuli are usually interfered with to a considerable extent; the corectopia may be unilateral or bilateral. Its combination with ectopia of the lens is very commonly observed; in such cases the condition is symmetrical and bilateral, and the iris is often tremulous. Sometimes gross developmental changes are present as well. An excellent synthesis of our knowledge on the subject will be found in a paper by Best [1].

Of these different varieties it is unnecessary to speak further; they are, with the exception, perhaps, of the first group, associated with changes in peripheral structures of an unmistakable kind.

My attention, however, has been drawn to the occurrence of a very definite degree of ectopia pupillæ in certain mesencephalic lesions, to its establishment at a certain point in the course of the disease, and to its persistence thereafter as a symptom of central disturbance. Since it first came under my notice I have found ectopia pupillæ to supervene during the course of not a few cases of disease in the mid-brain, but I shall confine myself to the details of three cases—chosen because of the differences in the character and duration of the ectopia—in two of which the results of *post-mortem* examination are accessible. These cases are the more significant when it is remembered that any congenital anomaly, or the operation of any cause of an extraneous nature, such as old inflammation, can be entirely excluded, for the ocular condition was perfectly normal when the patients first came under observation and changed into one of corectopia during their stay in hospital.

Case 1.—C. V., aged 24 years, was admitted to the National Hospital under Dr. Ferrier, on August 17, 1904.

In March of that year he began to be easily worried, and noticed he was rather awkward when on his bicycle. A month later he had constant headache all over his head, was frequently sick, and developed diplopia. His eyes were examined then and found to be normal, while his vision was pronounced to be $\frac{6}{6}$ in each. By the middle of May the headache had become chiefly frontal, and at the same time his caligraphy was noticeably impaired. His gait soon became weak and uncertain, and he tended to fall to the right. He continued to suffer from severe vomiting. Not long before his admission he noticed that his speech was affected, and he began to swallow with difficulty. On account of increasing weakness he took to his bed, and the sickness became less distressing. In July he commenced to have difficulty in micturition, and the headache seemed to be concentrated over the left supraorbital region.

When he came under observation his vision was $\frac{6}{6}$ in each eye, the fundi were normal, the pupils regular, equal, and in the centre of the iris, and their reactions were unimpaired. There

was well marked paresis of the left external rectus, and to a less extent of the right. Nystagmus was readily obtained in all directions. The motor and sensory functions of the left fifth nerve were considerably impaired, the left face was weak in both upper and lower sections, while the movements of tongue and palate were normal. Articulation was slow but not defective, and deglutition was also slow. There was a considerable degree of loss of strengh in all voluntary movements of the right limbs, but no objective impairment of sensation beyond the facial hypalgesia already mentioned. Ataxia of the right limbs was very obviously present, and the deep reflexes on that side were exaggerated, with ankle clonus and an extensor response.

At first the patient improved, and the facial sensory changes disappeared, leaving only corneal anæsthesia behind. In January, 1905, however, the left palate became paretic, and during the first week of February optic neuritis set in rather rapidly. By the end of the month his headaches became almost unendurable, the dysphagia and dysarthria increased, and he began to have attacks of respiratory embarrassment. Re-examined on March 25, he was found to have double sixth paralysis, considerable limitation of upward and downward movement, and five dioptres of swelling in each disc. Pupillary reactions were still normal, but the pupils were slightly deviated, bilaterally and symmetrically, in the direction of the upper and inner quadrant. The functions of the left seventh and eighth nerves were markedly impaired, the palate immobile, the whole of the right side of the body in a state of flaccid palsy, and hypalgesic, with the exception of the right face. The plantar reflex still gave a flexor response on the left side.

From this time the ectopia pupillæ became quite obvious and invariable, and though the iris continued to react sluggishly in the whole of its circumference under the stimulus of light, it was not sufficient to obliterate the ectopia. On April 16 the patient died of respiratory failure.

At the *post-mortem* examination it was seen that there was a large lobulated tumour of a white colour on the ventral aspect of the pons, extending externally from the upper part of the medulla to the points of exit of the third nerves. Laterally it spread into the cerebellum on both sides. Along the middle line, where the vertebral artery lay, the growth was heaped up on either side, with a deep channel in which the artery coursed. In one place it was

FIG. 1.

FIG. 2.

FIG. 3.

FIG. 4.

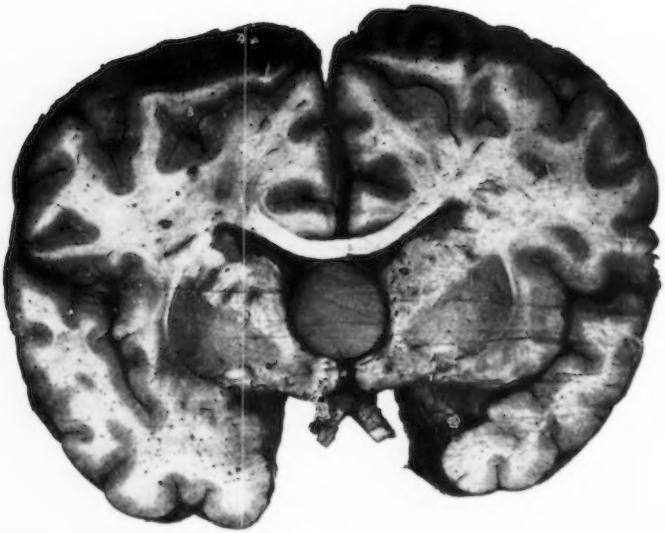


FIG. 5.

completely bridged over by the tumour. Both third nerves were flattened over the tumour; so were the fifths. On both sides the sixth cranial nerve was embedded in the tumour, the right one being overgrown in more than one portion of its extent. The seventh and eighth were between the tumour and the cerebellum, while the ninth and tenth passed out through the substance of the tumour. The eleventh seemed normal on both sides.

A section through the mid-brain (see fig. 4) at the level of the oculomotor nuclei shows how greatly the normal arrangement of the parts has been disturbed. The corpora quadrigemina are flattened; the gray matter round the aqueduct is quite altered in shape, being spread out laterally; the lumen of the aqueduct itself is no longer vertical, but has become a sinuous cavity, the anterior and posterior walls of which are almost in apposition. Upon the nuclei forming the oculomotor group there has been considerable pressure, whereby they have been distorted and are with difficulty recognised. In the corresponding nerves some degenerated fibres are discernible. The inner part of the left crus is involved in tumour growth, but there is no indication of spread of the actual tumour in a ventral direction.

Case 2.—G. S., aged 42 years, was admitted to the National Hospital, under Dr. Beevor, on October 19, 1904.

Three months before he suddenly had a severe bout of headache and vomiting, and after half an hour fell down unconscious. He soon recovered, but from that time suffered from constant headache, with occipital and frontal pain. Before admission he had in all about eight of these fainting attacks, which were preceded by an aura of giddiness and usually lasted for five minutes, though sometimes they were very protracted. Apparently there were no convulsive phenomena. Otherwise, he felt quite well.

On examination there was no indication whatever of organic disease of the nervous system, beyond the fact that the left pupil was slightly larger than the right, and, while both contracted well on accommodation, their reaction to light was sluggish. An attack occurred on November 27, and he was carefully observed through the whole of it.

For some hours previously he was more or less stupid and dazed, and vomited twice without apparent cause. In addition,

he was incontinent of urine several times. Towards evening he became to all appearance unconscious, his eyes closed, and the right side of his body began to tremble. On lifting the eyelids it was seen that the pupils were both small, and that there was marked external strabismus in each eye. He remained in this condition for about twenty-four hours, and, when it came to an end, the visual axes remained divergent, and there was slight double ptosis. The pupillary reactions to stimuli were normal.

At intervals during the next few weeks these attacks recurred. At 1 p.m. on December 13, for instance, it was noticed that he was disinclined for dinner, and sat gazing at it in a dreamy way. At 2 p.m. he vomited. By 4 p.m. he was almost unconscious, and the right corneal reflex could not be obtained. He moved his limbs about aimlessly, and occasionally put his hands to his head. The pupils were of medium size and reacted very little to light. He was incontinent of urine. At 7 p.m. he was totally unconscious; his pupils were dilated and did not respond to the stimulus of light, the eyeballs were markedly divergent, and the right limbs were flaccid, while the left were somewhat stiff. There were no tonic or clonic movements of any kind.

During the night he improved somewhat, and by the next morning he was himself again.

From that time, definite ophthalmoplegia was noted. Upward movements became very defective, on convergence only the right eye moved in, conjugate deviation to the left was limited and the ptosis became more obvious. In addition, a certain amount of proptosis was remarked.

On January 23, examination of the optic discs showed that papillitis had set in on both sides.

In the course of the next attack of unconsciousness it was noticed that both pupils were deviated from their former central position in the iris, and had moved upwards and inwards. This ectopia pupillæ persisted during some hours, during which time the pupils scarcely reacted to light at all.

As soon as the attack was over, the abnormal condition of the pupils disappeared.

On February 24 he woke at 6 a.m. feeling sick and dazed, and at 7 a.m. he vomited and was incontinent of urine. An hour or two later his pulse was 50 per minute, and he vomited again. By 10 a.m. he was absolutely unconscious, the corneal reflexes were abolished, his respiration was deep and stertorous, his face cyanotic, his arms extended and rather rigid. When his eyes were examined at 11 a.m., there was well-marked ectopia pupillæ

in each, the iris in the upper and inner quadrant being about one half as broad as the rest. The outline of the pupils was regular, and they were practically equal, but quite immobile to a strong light. He lay extended on his back, and ankle clonus and an extensor response were readily obtained on both sides.

At 5 p.m. no change had taken place. At 6 p.m. his pulse was 116, his temperature 99·6; the ectopia continued, and the conjunctivæ were somewhat injected. The legs were extended and rather flaccid, and neither ankle clonus nor extensor response could now be elicited. At 7 p.m. it was noticed that inspiration was "sobbing" in character, consisting of several separate inspiratory efforts, followed by a long expiration. The respiratory embarrassment continued, became aggravated, and shortly after 10 p.m. he died.

Post-mortem, an almost circular, or rather spherical, tumour, about the size of a large marble, was found occupying and greatly distending the third ventricle (see fig. 5). A cross section at its widest diameters measured 25 by 24 millimetres. It appeared to be growing from, as it was attached to, the ependyma of the ventricle. There was very considerable distortion of the surrounding parts, the pressure in all directions evidently having been severe. The floor of the ventricle was flattened out, the corpora mamillaria being compressed; the optic tracts had suffered, as well as the tip of the temporo-sphenoidal lobe, which was softened on both sides. There was no indication of actual invasion of the upper part of the pons, or of the gray matter round the aqueduct, but pressure downwards and backwards had dilated the latter somewhat, and flattened the former. The tumour itself was peculiarly homogeneous and colloid-like, of firm consistence and encapsuled in a thin membrane.

Case 3.—S. N., aged 32, was admitted to the National Hospital, under Dr. Taylor, on March 8, 1905.

Three months previously, she noticed the gradual onset of tingling in the feet, passing up the legs to the knees, and at the same time the legs felt heavy and weak. A month later she began to stagger as she walked and a similar tingling, with accompanying weakness, was remarked in the right hand and arm. Two weeks before her admission, she suddenly lost power in the legs almost completely, and soon after became unable to raise her arms above her head.

When she came under observation, there was no impairment of any of the cranial nerves, except that the facial musculature was weak and the features expressionless. The pupils were of medium size, regular and equal, with normal reactions. In comparison with the left, the right optic disc had a filled-in appearance. There was general weakness of the arm and leg muscles, especially the latter. On the whole of the left half of the body, with the exception of the face, there was slight diminution to tactile and painful stimuli; the deep reflexes were exaggerated, and ankle clonus and extensor responses were easily obtained on both sides. The sphincters were normal.

One month later, the general weakness of the musculature had greatly increased, and sensation was considerably diminished over the whole of the body as far as the shoulders. Further, the patient had difficulty in initiating the act of micturition.

During April, 1905, she became still worse. Wasting of the small muscles of the hands was noted, with considerable wasting of the muscles of the legs. The latter were very weak indeed, whereas the range of movement of the arms was still rather better.

On the evening of April 23, for the first time, and again at 11 a.m. on the 24th, it was noted that there was a certain amount of ectopia pupillæ on both sides, the pupil being deviated upward and inward. The pupils were regular, equal and of medium size, and reacted quickly both to light and on convergence.

At 3.30 on the same afternoon, the right pupil was distinctly larger than the left (5 and 3 millimetres diameter respectively), and the ectopia was more pronounced in the former. Its reactions were still good. On the morning of the next day, the patient remarked that she saw double on looking downward, and on the evening of the same day the right eyelid had begun to droop.

Examination on the 27th showed that there were ptosis and slight external strabismus on the right side, with some impairment of ocular movement, suggesting an oculomotor paresis. The following is a transcription of the note I made at the time:—

"When patient is asked to look up, both eyes move synchronously until each is in a position midway between the horizontal plane and the upward limit, when a fine nystagmoid movement begins in the left and is continued as the eye moves up to the normal extent; the right eye remains at this level, deviating slightly out and becoming quite stationary.

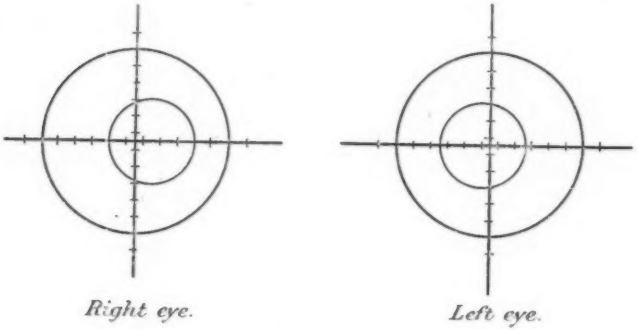
"Downward, both eyes move rather jerkily, but, while the left

moves to the usual extent, the right ceases to move when the lower margin of the pupil just reaches the margin of the lower lid.

"Conjugate deviation to the left is jerky—'punctuated,' to use Dr. Hughlings Jackson's expression—and fine quick nystagmus begins when the eyes are halfway between the mid and extreme positions. The right eye does not move further into the inner canthus, remaining with the whole of the inner iris, and some of the conjunctiva, quite visible.

"Both eyes move well and synchronously to the right, and a slow, fine nystagmoid jerking is seen as they approach the extreme.

"Convergence remains fairly good, the movement of the right



CASE 3.

Each division on the vertical and horizontal lines represents one millimetre.

[When the measurements were taken the right pupil was deviated rather more upwards than is indicated in the diagram.]

eye being possibly rather better than on combined movement to the left."

In the right eye, the corectopia was well marked, and its measurements in millimetres are indicated on the accompanying diagram. The pupil was now rather less in size, but still larger than the left; in the latter eye the displaced position of the pupil was quite definite, though not so pronounced as in its fellow. Both reacted to light, less on the right than on the left, and on convergence; further, they both dilated to shade. On examining with a lens it was easy to see that the iris was "condensed," so to speak, in the upper and inner quadrant; *i.e.*, the radial projecting ridges were much closer together than in the rest of the iris, and the corresponding portion of the pupillary zone was

narrower and appeared thicker. The outline of each pupil was almost regular. In neither optic disc could any ophthalmoscopic appearance be found that was recognisable as connected with central nerve disease.

Thereafter, the ophthalmoplegia became more marked for some days, with constant diplopia, but improvement soon set in, and after about a month no sign of preceding palsy could be discovered, though there was still nystagmus on lateral movement in either direction. The ectopia pupillæ was less marked, but it was still more obvious in the right than in the left, while the shape of the pupils was regular, their size equal, and their reactions normal.

A visit to the patient six months later allowed me to satisfy myself that there was merely a trace of a corectopic position on the right and left sides, and nystagmus on lateral deviation, but no other pupillary or ocular anomaly.

To recapitulate in a few words the salient features of these cases, the details of which have been given :—

In the first, double symmetrical corectopia supervenes in the course of a pontine tumour at least four weeks before the patient's death, and continues till the end. Clinically there is considerable impairment of ocular movement, and pathologically there is evidence of involvement of the oculomotor nerves, among others.

In the second, a patient with indications of intracranial growth has peculiar congestive attacks in which he becomes unconscious for hours, during which ectopia pupillæ is remarked on both sides. It disappears after one of these attacks, but reappears during the final one, which has a fatal termination. In his case also there is ophthalmoplegia, and a tumour is subsequently discovered occupying the whole of the third ventricle, and pressing down and back on the upper part of the pons.

In the third, which can scarcely be other than disseminated sclerosis, ectopia pupillæ appears during the patient's sojourn in hospital, when there is definite defect of ocular movement, and about thirty-six hours before the development of an incomplete oculomotor palsy. It continues for a month or more, and improves, though it does not entirely disappear, with improvement in the other ocular symptoms.

What have the cases in common?

It may be remarked at once that in no one was a history of syphilis forthcoming, and in two of them the operative cause was shown to be something else than syphilis. Further, it is abundantly clear that any congenital anomaly, or any sign of fœtal inflammation, can be discounted; for, when the patients came under observation originally, their pupillary condition was absolutely normal. Moreover, in no one of the series was there ever any indication of accompanying iritis or other peripheral disturbance, such as might conceivably have effected the pupillary displacement.

We are accordingly compélled to s‹ ek another explanation of the corectopia, viz., in the central lesions which the cases present.

A brief reference may, with advantage, be made at this point to the anatomy of the iris and the physiology of pupillary movement, as far as these are at present established.

The pupil constrictor fibres, which arise from the nucleus of the oculomotor nerve in the floor of the aqueduct, run in that nerve till the orbit is reached, when they pass into the twig supplying the inferior oblique, and so by the short root of the ciliary ganglion, the ciliary ganglion, and the short ciliary nerves to the eye. The pupil dilator fibres, which probably arise in the neighbourhood of the third nerve nucleus, pursue a course *viâ* the cervical sympathetic to the cervico-Gasserian strand and the Gasserian ganglion, thence by the nasal branch of the ophthalmic division of the fifth to the long ciliary nerves, thus avoiding the ciliary ganglion, and so to the eye.

Alterations in the size of the pupils may be the result of changes in any or all of three mechanisms, viz., the sphincter, the dilator, and the vaso-motor. In the iris are radially-arranged vessels, dilatation of which will produce some constriction of that structure, and constriction of which will dilate the pupil.

There is reason to believe that all three come into operation, though unequally, when changes take place in the pupil. Yet over the part that each plays discussion is still raging. It is undesirable and unnecessary, in the present com-

munication, to enter into detail where there is still so much difference of opinion. We may abide by the statement of Parsons that constriction of the iris may be due to (1) contraction of the constrictor muscle; (2) relaxation of the dilator muscle; (3) dilatation of the blood vessels. Dilatation of the iris may similarly be due to (1) contraction of the dilator muscle; (2) relaxation of the constrictor muscle; (3) constriction of the blood vessels, and that the three causes are effectual in diminishing degree in the order named.

The next point is, what is the mechanism of the form of ectopia with which we are immediately concerned?

In this connection we must refer to the researches of Piltz [5], Marina [3] and others on the relation between irregularities of outline and excentricity of position of the pupils and pathological changes in the cells of the ciliary ganglia. Marina found signs of degeneration in the ciliary ganglia and short ciliary nerves in every case of tabes or general paralysis that he examined which showed the characteristic pupils. Piltz exposed the ciliary ganglion and ciliary nerves in the dog, and by irritation of various twigs of the latter produced irregularities in the outline of the pupil and in its position. Hensen and Volkers [2] had already demonstrated that excitation of single short ciliary nerves caused local constriction, while section caused local dilatation.

The importance of these observations is of course great; but that the explanation of the corectopia we are considering is to be sought in alterations in ciliary ganglia and nerves can scarcely be admitted. In our second case, for instance, the possibility of the ciliary ganglia being influenced by a tumour of the third ventricle is indeed remote. In the first they were not removed for examination, it is true, but there was no indication of any spread of the tumour in the direction of the orbits, and any change in the ganglia could have reached them only by their motor or sensory roots. Moreover, in the cases here recorded, however excentric the pupils they did not present the irregularities produced experimentally by Piltz. It is to be remarked that in them all the deviation was in the direction of the upper and inner quadrant, and more or less symmetrical, whereas the deviation

secondary to changes in the ciliary ganglia is conspicuous by its irregularity. A conceivable connection between this more or less symmetrical ectopia and convergence of the eyes is negatived by the fact that in the second case the ocular axes were divergent.

It is more plausible to suggest that there is some relation between it and the disturbances of the upper part of the pons, which was a feature of all the cases. A glance at fig. 4 shows how the oculomotor nucleus area was distorted in Case 1; a not dissimilar condition obtained in the second; and in the third the corectopia persisted during an oculomotor palsy, and improved with the latter. Whether it is to be attributed to a segmental contraction of the dilator, or a segmental relaxation of the sphincter, or a segmental vasomotor effect, is a problem difficult of solution. The view I am inclined to take is that the last of these plays the chief *rôle* in its production. In all the cases the reactions of the pupil to ordinary stimuli were only slightly impaired, or not at all—with the exception of the second, but we must remember that the patient was comatose when the ectopia was observed. There cannot, therefore, have been any gross paralysis of the intrinsic muscles of the eye; even in the second case there was a return to the normal after the congestive attack was at an end. In testing with a bright light, the iris appeared to contract unequally round its circumference, but of this one could not be absolutely certain; in any case, though the corectopia became less obvious, it did not disappear. On the other hand, both cocaine and atropine acted on the pupils in their usual way; but the displacement of the latter was not entirely overcome. These facts seem to indicate that while there may have been partial paresis of one or other of the more important mechanisms, the vaso-motor one was involved in a greater degree. The dilator and constrictor fibres alike, as has been already remarked, arise in the mesencephalon in the neighbourhood of the third nucleus, and are probably most anterior in the oculomotor group. Hence they are perhaps specially prone to suffer where ventricular distention is great, as it was in the two first cases. Be this as it may, the suggestion is that the ectopia pupillæ of the series of cases detailed above is a

vaso-motor effect, since there was no gross interference with pupillary reactions, and that it is due to disturbance in the function of vaso-dilator and vaso-constrictor fibres which are closely connected in their central origin. The relation of vaso-motor fibres to the nerve fibres supplying sphincter and dilator is undetermined, though it is certainly an intimate one. The facts of Case 3 may suggest that a vaso-motor effect may precede, and be the first indication of, an oculo-motor disturbance; complete paralysis of either sphincter or dilator muscle will prevent the vaso-motor anomaly from being noticed.

I am conscious of the tentative nature of this proffered explanation of a condition interesting in itself, and instructive in so far as it leads to wider questions, *e.g.*, the segmental innervation of the iris, and its central connections. As has been impressed on me by experience, it is easy to miss the condition in a cursory examination, but once one is on the look out for it, examples seem to multiply. A search through recent text-books and literature for instances or references has been entirely unproductive, so that in briefly drawing attention to the subject one may be allowed to express the hope that subsequent observations will aid in the elucidation of its pathogenesis and clinical significance.

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Description of Plates.

Figs. 1 and 2.—Case 1.

Fig. 3.—Case 3. The pupil has contracted slightly as the patient was made to face the light for the photograph; the ectopia is therefore rather less noticeable.

Fig. 4.—Section through the mid-brain of Case 1 at the level of the oculomotor nuclei. The structures are all considerably distorted. Tumour growth has invaded the pyramid on the left.

Fig. 5.—Vertical transverse section of the brain of Case 2, showing the tumour occupying the third ventricle.

THE GROUPING OF AFFERENT IMPULSES WITHIN THE SPINAL CORD.

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APPENDIX.—Reports of the Cases Cited in this Paper.

Chapter I.

Introduction.

It would seem reasonable to suppose that the mechanism of sensation was arranged in such a way that, from the skin to the sensorium, there was one system of end-organs, fibres and cells for each sensory quality appreciable by man. On this supposition, every application of heat to the skin would cause a disturbance in the activity of a special set of end-organs, which reacted to this form of stimulation only. From these end-organs the impulse would be transferred by specialised fibres to the spinal cord, and thence by a single tract, directly or through the intermediation of cell-systems, to the final centres in the brain. On this hypothesis, similar organs must exist for cold, for pain, for pressure and for touch. With the discovery of the hot and cold spots, and with von Frey's [10] further development of the doctrine of punctate sensibility, a mechanism seemed to have been found which would reasonably explain the phenomena of sensation.

But recent work has shown that, as far as the peripheral nerves are concerned, the afferent nervous system is not arranged on so simple a plan. Rivers [23], in conjunction with one of us, has been able to show that between the sparsely scattered hot spots lies some other mechanism, capable of responding to stimulation by warm objects. Similarly, the work of Sherren [13] upon divided peripheral nerves has demonstrated the existence of two afferent systems in the skin, each of which has its own distribution. To explain the nature and distribution of the sensory mechanism in the peripheral nerves, we are compelled to fall back upon a hypothesis of evolution, from a simple apparatus to something higher but still imperfect; we cannot assume the immediate development of a system logically and specifically complete.

The conception, which we have reached, of the afferent mechanism in the peripheral parts of the nervous system, accords more clearly with what might be expected from our present knowledge of comparative anatomy and physio-

logy. For it is extremely unlikely that, out of the primitive sense-organs of lower animals, independent systems could be developed in man, specifically fitted to receive all those impulses which lead to the development of the sensations of heat, of cold, of pain, of touch, or of pressure. But our doctrine of three peripheral systems developed in all probability at different phylogenetic periods, each system reacting differently to the mass-stimuli of daily life, accords better with the known processes of evolution.

We concluded from our examination of the phenomena which follow division of peripheral nerves, that the afferent mechanism at this level of the nervous system consisted of three systems :—

(1) A system corresponding to the group of impulses we have called deep sensibility. The end-organs of this system respond to the stimulus of pressure and to the movement of joints, tendons and muscles. Painful impulses can also arise within this system in consequence of injury to a joint, or excessive pressure. This sensory mechanism is capable of responding in such a way that the patient appreciates both the locality of the stimulus and the direction of movement in any joint which lies within an area innervated solely by this system; and yet the integrity of deep sensibility carries with it no power of appreciating a stimulus, such as that of cotton-wool, even over hairy parts. Nor does it permit of the discrimination of two compass-points applied simultaneously to the skin, even when widely separated.

The fibres which conduct these sensory impulses run mainly with the muscular nerves, and are not destroyed by division of all the sensory nerves to the skin.

(2) The protopathic system, capable of responding to painful cutaneous stimuli and to the more extreme degrees of heat and cold. Its end-organs are grouped in points on the surface of the body, sensitive to one only of these stimuli. Their response is diffuse, and unaccompanied by any definite appreciation of the locality of the spot stimulated.

(3) The epicritic system. To the impulses of this system we owe the power of cutaneous localisation, of discriminating

two points and of recognising the finer grades of temperature, called cool and warm.

A knowledge of this grouping of afferent impulses was gained by studying the consequences which followed division of peripheral nerves and posterior nerve roots in man. But, on comparing the loss of sensation produced by injury and disease of the spinal cord with that caused by division of peripheral nerves, fundamental differences at once became apparent. For if, in consequence of disease of the spinal cord, sensibility to the pain of a prick was lost, the pain caused by excessive pressure was also abolished, even though the parts remained otherwise normally sensitive to pressure. Similarly, when a part became totally insensitive to stimuli which usually evoke a sensation of warmth, it was found to be also insensitive to all temperatures which normally cause a sensation of heat.

It is evident, therefore, that, as soon as a sensory impulse reaches its first junction in the spinal cord, it becomes shunted into tracts devoted to the conduction of impulses, grouped in a way different from that found in the peripheral nerves. It is no longer a question of protopathic, epicritic and deep sensibility; each tract in the central nervous system is devoted to the conduction of one of the specific impulses, such as pain, touch, heat and cold.[1]

In the present paper, we are concerned more directly with this grouping of sensory impulses in the spinal cord, rather than with the position of the tracts by which they pass to the higher centres. We hope to demonstrate, not only that a change takes place in the grouping of the afferent impulses, but also to show at what point this transformation occurs.

Methods.

The cases quoted in this paper occurred mainly in the practice of the London Hospital, but we have included some instances from the private practice of one of us. Most of the patients have been under observation for long periods, in some instances for over five years, and we have thus been able to gain an idea of their temperament and trustworthi-

[1] For the nature of the posterior columns *vide* Chap. VII., p. 636.

ness. For it must be remembered that observations, extending in many instances over large areas of the body, require the exercise of much greater patience and exactitude than the shorter examinations of the smaller areas of loss of sensation produced by injury to peripheral nerves. In many instances, we have rejected all our observations on a patient on account of his unsatisfactory mental state. Moreover, fatigue plays a considerable part in the answers obtained to such tests as we have applied, and we have therefore as far as possible made our observations in short but frequently-repeated sittings.

As far as possible, our observations were made in a quiet room, apart from the hospital ward with its distracting sights and sounds. When it was necessary, in consequence of the difficulty in transporting the patient, to carry out our tests in a ward, his bed was carefully screened and every method adopted to secure his undivided attention. In all cases the patient was in bed, and the parts to be tested were exposed as little as possible. In the winter, the accuracy with which replies were given over parts of diminished sensibility was, as a rule, less than in the summer. This is not altogether a matter of external temperature; for it was found that a damp, misty or foggy day was peculiarly unfavourable for testing sensation. Anything that produces a feeling of coldness, anything which causes shivering or the appearance of "goose skin," greatly diminishes the accuracy of the answers given to most tests. The most favourable conditions are a warm day of early summer, or a bright cool winter morning in a well-warmed room.

It is important that the patient should be free from all visceral discomfort; he must not be hungry, nor suffer from a desire to empty his bladder. During the testing of R. A. H. (Case 16) it was noticed that his answers became much less accurate than they had been earlier in the day; for, although the right forearm was sensitive to touch and at any rate not totally insensitive to a prick, he failed eight times out of twenty attempts to tell the head from the point of a pin. He was then allowed to empty his bladder, and from that time made no further mistakes. His answers, which had shown much confusion, were now uniformly correct.

During the testing, the patient's eyes were closed; or in some instances, especially where the area under observation lay over the lower extremities, he was allowed to lie with his eyes open, but a screen was placed across the bed so that he was unable to see the hands of the operator or the tests to be applied. Some patients are more comfortable when this system is adopted, than if they are forced to remain for long periods with closed eyes.

Superficial touch was tested by means of cotton-wool, stroked gently across the part. Unfortunately, this stimulus, so perfect a test over parts endowed with hair, is untrustworthy over hairless parts, unless used with extreme caution. Many brands of wool, when rolled into a wisp, form so stiff a mass that deep sensibility (pressure) is evoked; but if this cotton-wool is frayed out, so that it can evoke sensations of light touch only, it frequently fails to stimulate the horny palm of a workman, or even some parts of a normal, well-kept hand.

In some instances we have used a set of hairs, kindly prepared for one of us by Professor von Frey. Each consists of a hair of different resistance to bending, fixed to a small wooden rod at a right angle. These hairs are calibrated by determining upon a small balance the pressure necessary to bend them. We wish to protest against a common variation of von Frey's apparatus, which consists of a single hair in a metal sheath, so arranged that it can be protruded or withdrawn to a varying extent. The condition of such a hair varies greatly, and the pressure necessary to bend it varies from time to time, even when it is extruded from its sheath to the same amount. Moreover, in order that the hair may not suffer by the extension and withdrawal, it must of necessity be thicker and coarser than when the test is made with a series of hairs, each permanently affixed to its own handle.[1]

[1] The actual hairs used by us, which we owe to the kindness of Professor von Frey, bent at the following pressures: No. 8 at 830 mgm., No. 5 at 360 mgm., No. 4 at 230 mgm., No. 2 at 100 mgm. These hairs are kept in a box with the handles supported on a rack so that the hair remains always free from contact. The force required to bend them varies slightly according to use and the condition of the atmosphere; but after nearly four years No. 8 still turns the scale at 800 mgm., No. 5 at 320 mgm., No. 4 at 210 mgm.

The power of discriminating two points was tested with a pair of compasses, the ends of which had been blunted. The method adopted differs from that in general use, and is an amplification of that suggested by McDougall.[1] The compass-points were set at a certain distance from one another; they were then applied to the part to be tested in such a way that sometimes two points, sometimes one point only touched the skin. No questions were asked, but, before the testing began, the patient, whose eyes were closed, was told to say with every application how many points he perceived. The stimuli followed one another in an entirely irregular order, but so that, ultimately, the patient had been touched ten times with one point, ten times with two points. Each series was recorded in such a manner, that it became immediately obvious in how many cases among the ten single, and among the ten double stimuli, the patient had given a wrong answer.

In some of these experiments it was impossible to compare the results obtained over an abnormal part with a similar part of normal sensibility. When we were examining the consequences of injury to a peripheral nerve of the hand, we always compared our results with those obtained from the normal hand. But in the patients used for the present research, the opposite extremity was not infrequently in a condition of abnormal sensibility. We have therefore paid no attention to fine differences in precision, and, as will be seen from a perusal of our results, have dealt with large differences only.

The affected parts were also tested with a tuning-fork, and for this purpose we have used one beating 128 double vibrations a second. This is a treacherous form of stimulation, unless the patient is intelligent and the observer constantly on his guard. Error not uncommonly arises in consequence of conduction of the vibrations from an insensitive to some sensitive part at a distance; the patient then appreciates the stimulus, but not at the spot to which the fork was applied. It is most important that, among every set of stimulations, the fork should be from time to time

[1] It has been fully described in BRAIN, 1905, p. 228.

applied when not vibrating, as patients occasionally answer "yes" when they appreciate only the pressure of its flat base, and not that sensation of vibration which is the essence of this test.

Ability to recognise objects of different sizes was tested by means of circular steel discs with diameters of 2 cm., 0·75 cm., 0·2 cm. Of these, either two or three were applied indiscriminately to the part one after the other, and the patient said "The first was larger than the second and the second was smaller than the third." If care was taken to apply these discs to the skin with approximately equal pressure, this simple method was found to work well in practice and gave better results than more complex procedures.

We have obtained no satisfactory results in pathological cases from any tests we have used to determine the power of appreciating the shape of an object applied to the skin. Zinc, indiarubber, wood and cardboard cut into simple figures, such as squares, circles and crosses, even when made to 5 cm. in diameter, could not be appreciated over the normal skin with sufficient certainty to be useful for clinical examination.

Sensibility to one form of pain was tested by means of a needle, or sharp pin, in the usual way. But, in dealing with patients suffering from some lesion of the spinal cord, in whom sensibility to touch is frequently unaffected, it is particularly necessary to be certain that a correct answer ("prick") is accompanied by the appreciation of pain. For most of these patients can discriminate stimulation with the point from a touch with the head of a pin, although totally insensitive to the pain of a prick. This acuæsthesia is, in reality, a form of the appreciation of size, and is closely associated with the integrity of touch. If, for this reason, there was any doubt concerning the patient's power to appreciate the pain produced by a prick, a painful interrupted current was used as the test, and by this means it was easy to discover whether the patient's answers were based on the perfection of his acuæsthesia or upon a true appreciation of pain.

The interrupted current can be used to test sensibility to

touch or to pain, as was pointed out in a previous communication [13]. In this research we have used it mainly as a painful stimulus, and on such parts only as the palm have we employed the painless interrupted current.

Fortunately, fewer mechanical difficulties surround the testing with heat and with cold when the lesion lies within the spinal cord, than after injury to a peripheral nerve. For, if sensibility to either heat or cold is disturbed in consequence of an intramedullary lesion, the appreciation of intermediate degrees is disturbed, together with that of the extremes. We have, therefore, usually employed glass test-tubes, but in some of our experiments have used silver tubes for the more delicate observations.

It is well to remember, when testing the scalp, that the hair insulates the skin, so that both heat and cold pass through with difficulty, and the results are liable to be unsatisfactory.

As tests for the extremes of heat and cold we have used ice and water at 50° C. The temperatures selected for the intermediate degrees varied with the external temperature, and were in every instance applied to some normal part, such as the forehead of the patient and of the observer. Moreover, control tests were always made with a tube of a neutral temperature, neither hot nor cold.

One of the greatest difficulties, familiar to all who have investigated cases of diseases of the spinal cord, is the tendency of the patient to call all temperature-stimuli, whether hot or cold, by the same name. It is most important, under such circumstances, to interject frequent stimulations with a test-tube which is neither hot nor cold to the normal skin. Then it will be found that, of the two stimuli, the neutral tube is as frequently said to be hot or cold, as the tube which is hot or cold to the normal skin.[1]

The existence of a sensation of pressure was tested by means of a pencil, or other blunt object. After division of a peripheral nerve to the skin, whenever pressure is appreciated, its steady increase leads to the production of pain. But when the lesion is situated within the spinal cord this

[1] For one source of this fallacy, see p. 681.

is not the case, and it therefore became necessary to possess some means of measuring the amount of pressure capable of causing pain. For this purpose we have used a modification of Cattell's algometer, made for us by the Cambridge Scientific Instrument Company. The apparatus, as made in New York, consists of a cylindrical rod with a diameter of ·75 cm., which runs into a large handle. Pressure upon the free end of this rod compresses a spring, and the amount of this pressure can be read off from the movements of a marker on a scale let into the handle. The observer holds the instrument in his hand and presses the free end of the rod on to the skin of the patient, who cries out as soon as pain is produced. The amount of pressure applied is read from the height of the marker on the scale. But we found that this instrument was liable to give faulty readings, due to mechanical difficulties in constructing an inverted balance that will read correctly. The modification we have used was suggested by Dr. Rivers, who superintended the manufacture of our instrument. (Figs. 1 and 2.) There is no scale in the handle, which contains only the spring to be compressed. But on the rod slides a scale so arranged that, when pressure is made and the rod is driven into the handle, the scale is pushed down upon the shaft of the rod.

Immediately the patient calls out that the pressure has become painful, the instrument is removed, the rod springs out of the handle again, carrying with it the scale, which remains at that point where the rod emerged from the handle at the moment of maximum pressure. A line drawn around the rod acts as an indicator, and the amount of pressure applied can be read off at leisure from the relation of this line to the measure on the scale. This scale is graduated in kilograms.

An additional advantage obtained from this method of registration, is the ease with which the spring can be made to give constant readings. Such constancy is extremely difficult to obtain, if the spring is fitted with the necessary apparatus for obtaining readings by means of a marker on a scale.

We have found an algometer, constructed with these

FIG. 1. FIG. 2.

FIG. 1.—Shows the Algometer before use. The zero of the scale corresponds to the horizontal line on the rod.

FIG. 2.—Shows the Algometer after use. The horizontal line on the rod now corresponds to 11·5 divisions on the scale. At this point the patient complained that the pressure caused pain. The graduations correspond to kilograms.

modifications, of the greatest use. It will give different readings in the hands of each observer on account of variation in the manner and rapidity with which the instrument is applied. We therefore neglect all small differences, paying attention only to those so gross that it is certain there must have been some grave defect in the patient's power of appreciating the pain of pressure.

We employed the following method for testing the power of recognising passive movements, and the position into which a limb had been placed passively. After closing the patient's eyes, the thumb, finger or great toe is grasped by the observer in such a way that his forefinger and thumb press on its lateral aspect. Then the finger or toe can be flexed or extended to a varying degree, without applying pressure in the direction of movement. When the wrist is to be flexed or extended, the movement is produced from one of the fingers, the arm being supported, so that the hand moves freely. Movement of the elbow is made by grasping the wrist laterally, and the knee is tested by supporting the thigh and flexing or extending the knee from the ankle. Thus every joint is moved by means of the one distal to it.

The patient is asked to say what movement is made and how the part under examination lies when movement is over. But we lay much more stress upon the results of the following test. He is asked to imitate the movement with the limb of the opposite side. For instance, when the thumb of the right hand is under examination, every flexion, extension, or other movement, passively produced, should be followed by a similar voluntary movement of the thumb of the left hand. When both legs are affected, as is so commonly the case, in consequence of lesions of the spinal cord, the patient is asked to imitate with the thumb the movements of the great toe, and with the wrist those of the ankle.

Frequently, when in doubt, he will guess, and we therefore from time to time allow the part to remain quiescent in a definite position, still grasped between the fingers, after a movement has been made. Perhaps the answer given by

the patient has corresponded correctly with the movement made by the observer. But, in spite of the part remaining stationary in the same position, the patient indicates that it is being moved. This is one of the most certain signs, either that he is untrustworthy, or that there is some grave defect in his power of recognising position assumed passively. Occasionally, when the patient's answers have been incorrect, we allow him to open his eyes, and his look and exclamation of astonishment on seeing the true position of his limb is strong evidence that his power of appreciating the position of the part is gravely diminished.

It is well to adopt a method of recording the answers similar to that used for the results of the compass-test. Ten movements are made in one direction, ten in the other, indiscriminately, and the results are marked above and below a line, each answer being recorded by a stroke if right, by a cross if incorrect. If no answer is given, an O is inserted, whilst doubt is indicated by a D. We look upon this method as greatly increasing the accuracy of the record. (*Cf.* the records on p. 660).

Many methods have been used for testing the certainty with which a patient can indicate the point touched. For the purposes of this research we have used the following methods.

First, the patient, whose eyes were closed throughout the whole period of testing, was asked to name the part touched, pressed or otherwise stimulated. Secondly, he was told to point to the spot stimulated. In the case of the legs, he was given a cardboard rod to be used as a pointer. His eyes remained rigidly closed throughout the whole period of testing ("groping method"). Thirdly, a hole was cut in the centre of a stiff sheet of paper, and through this he was stimulated, the paper being held about 1 cm. distant from the skin. He was given a soft pencil and with this tried to point to the spot stimulated. The point he reached was marked on the paper, and the distance from the central hole could be measured. (Spearman's method [31]).

In this research we have rarely used the method of Victor Henri [14], in which the patient is given a life-

sized diagram of the part to be tested and is asked to mark upon it the point stimulated. Such a method is of great value when the hand is affected, a condition which seldom occurred in the cases brought forward in this paper.

Chapter II.

The nature of the loss of sensation produced by a lesion of the spinal cord, compared with that due to division of peripheral nerves.

Afferent impulses pass from the periphery to the spinal cord along three systems of fibres, those of protopathic, epicritic, and deep sensibility.

The protopathic system is essentially one of punctate sensibility. For heat, for cold and, probably, for pain, there are definite spots, from each of which a specific sensory impulse can alone be released. The hairs are also innervated partly from this system; and, when the skin is endowed with protopathic sensibility only, exquisite pain is produced by plucking the hairs, and a curious diffuse tingling or formication occurs when they are brushed with cotton-wool.

Elimination of epicritic sensibility from any part of the body makes it impossible to distinguish two points of the compasses applied simultaneously, cutaneous localisation is perverted and light touch over hairless parts abolished. Whenever epicritic sensibility is destroyed, the power of appreciating intermediate degrees of heat and cold ("warm" and "cool") will also be lost. We believe that there is a separate cutaneous mechanism which reacts to temperatures we call warm (34° C. to 40° C.). It is also possible that coolness is due to impulses from end-organs other than the cold spots; the evidence for this view will form the subject of a further communication by one of us in conjunction with Dr. Rivers. But it is also conceivable that the sensation of "coolness" might be produced by the combined stimulation of this mechanism and the cold spots which

belong to the protopathic system. According to this view an appreciation of "warmth" would be necessary for the production of the sensation of "coolness," and therefore destruction of epicritic sensibility would remove the power of appreciating the intermediate degrees of temperature, whether they normally give rise to a "warm" or a "cool" sensation.

The existence of both these forms of sensibility depends upon the integrity of fibres in the sensory nerves to the skin. But, even after the complete division of all nerves to the skin, the denervated part remains sensitive to pressure, and pain can be produced as soon as this pressure exceeds a certain measurable amount. Each pressure-touch can be localised with fair accuracy, and any movement of the joints is at once recognised. But no degree of temperature produces any sensation, and the part may be burnt or frozen with impunity; not even the pain of the extremes of heat and cold can produce a reaction in this system, which is so exquisitely sensitive to the pain of increasing pressure. The two points of the compasses applied simultaneously cannot be discriminated, but the vibrations of a tuning-fork beating 128 to the second can be perfectly recognised.

As this form of sensibility remains after all cutaneous afferent nerves have been destroyed, it is obvious that the fibres by which its impulses pass to the central nervous system must run, at any rate to a considerable extent, with the muscular nerves. But it must not be supposed that we deny the existence of fibres in the superficial structures of the body, which belong to this deep system, and are capable of reacting to pressure in all forms.[1] Sherren, Rivers and Head were compelled to neglect them, because their only opportunities for investigating the properties of this deep system arose after division of all the nerves to the skin. But, in consequence of the peculiar dissociation of sensibility which we shall study in this paper, we see reason to believe that this system of afferent fibres running in great part with muscular nerves, also sends some fibres to the skin, particularly of such parts as the palm of the hand and the sole of the foot.

[1] *Cf.* BRAIN, 1905, cx., p. 292.

These statements can be summarised in the following table :—

Loss of *epicritic sensibility* abolishes—
- Recognition of light touch over hairless parts or parts that have been shaved.
- Cutaneous localisation.
- Discrimination of compass points.
- Appreciation of differences in size, including the accurate discrimination of the head from the point of a pin apart from the pain of the prick (acuæsthesia).
- Discrimination of intermediate degrees of temperature from about 25° C. to about 40° C.

Loss of *protopathic sensibility* abolishes—
- Cutaneous pain, especially that produced by pricking, burning or freezing, together with that of stimulation with a painful interrupted current. Over hair-clad parts, plucking the hairs ceases to be painful.
- Sensations of heat from temperatures above about 45° C.
- Sensations of cold from temperatures below 20° C.

After destruction of all cutaneous afferent fibres the part is still endowed with *deep sensibility*.
- Pressure can be recognised, and its gradual increase appreciated.
- Pain is produced by excessive pressure (measured by the algometer).
- Movements of muscles can be recognised.
- The point of application of pressure can be localised.
- The patient can recognise the extent and direction of movement produced passively in all the joints within the affected area.

This grouping of the afferent impulses on their way from the periphery to the central nervous system is probably determined by reasons of phylogenetic development. But as soon as the first intramedullary cell-system is reached, these impulses become shunted into paths along which they travel in new combinations, and in this chapter we shall discuss the forms assumed by the loss of sensation, due to a lesion interfering with afferent impulses, after this new grouping has occurred.

Section 1.—*Pain.*

Nowhere is this difference of grouping more evident than in the behaviour of those impulses associated with the pain produced by deep pressure. After division of a peripheral nerve, or of posterior roots, those parts only become insensitive to the pain of deep pressure which are at the same time totally insensitive to the tactile element of this stimulus.

Unless all forms of deep sensibility are abolished, pain will still be caused by excessive pressure.

But, if the lesion lies within the spinal cord, sensibility to pain is abolished as a whole, whatever the form of painful stimulation. The patient will be insensitive both to the pain of excessive pressure and to that of a prick or of the interrupted current; yet, at the same time, he may be able to recognise the position of the spot pressed, and can appreciate the gradually increasing pressure.

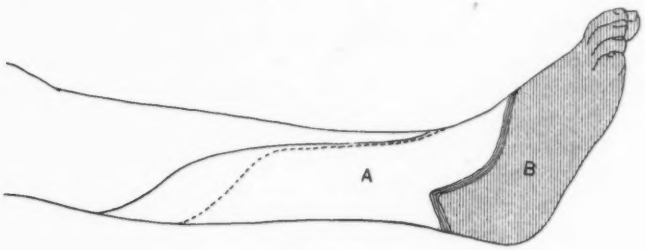


FIG. 3.

To show the loss of sensation in William B., produced by destruction of the sacral plexus.

The extent of the loss of all forms of sensation, including pressure, is shaded and marked **B**.

Those parts which were insensitive to all cutaneous painful stimulation but were sensitive to the pain of deep pressure are enclosed within a broken line and marked **A**.

Sensibility to light touch was lost below the single dark line.

The following case is an excellent instance of the condition produced by destruction of several nerve-trunks.

William B. (Originally reported by Sherren and Head [13], and later by Sherren [29]) was shot through the buttock on October 4, 1901. This produced loss of sensation over the region supplied by the great and by the small sciatic nerves, together with paralysis of all the muscles below the knee supplied by the latter nerve. He was seen by Mr. Sherren and one of us on several occasions from 1902 onwards. Finally, in May, 1905, he came under the care of Mr. Sherren at the London Hospital, and at the exploratory operation the following condition was found. The sciatic plexus was divided below the point at which it was joined by the second sacral nerve;

the third sacral had been destroyed in the wound of the sacrum and the nerves were bound up in a dense mass of fibrous tissue. Mr. Sherren sutured the lumbo-sacral cord and the second sacral nerve to the stump of the sciatic. As soon as the wound of this operation was healed, the sensibility of his leg was again tested and was found to correspond exactly to that before the operation. The extent of the loss is shown on Fig. 3, taken from a series of photographs. It will be seen that over the greater part of the foot, deep sensibility was lost (area *B*); here he was insensitive to the painful and tactile elements of pressure and failed to appreciate the vibration of a tuning-fork. But on the outer aspect of the leg was an area of considerable extent (*A*) over which he was entirely insensitive to the prick of a pin or to the pain of an interrupted current. Within this area, the same amount of pressure was painful as on the sound side within similar limits. Thus a part of the leg completely insensitive to cutaneous painful stimulation was sensitive to the pain of excessive pressure. The only parts where pressure could not be made to produce pain, were those devoid of all forms of deep sensibility.

	Algometer Reading in Kilograms.	
	R. (affected).	L.
Outer Aspect of Shin. Anterior portion (*a*) ..	Pain at 2 (within the area of cutaneous analgesia) ..	Pain at 2.
Posterior portion (*b*) ..	Pain at 3 (within the area of cutaneous analgesia) ..	Pain at 3.
Sole of foot	No pain at 10 (deep sensibility abolished)	Pain at 2.
Below external malleolus	No pain at 10 (deep sensibility abolished)	Pain at 1·5.

Compare with this dissociated analgesia the condition of the following patient (Case 17), who suffered from a lesion within the spinal cord. He was injured by the overturning of a truck of concrete on May 9, 1905, and was brought to

the London Hospital paralysed from the hips downwards. By May, 1906, he had partly recovered and showed a condition that was so sharp a contrast to that of the previous patient. (Fig. 4.)

He walked, dragging the left leg like a man with hemiplegia, and this leg was stiff and spastic. All movements could be performed by the various parts of the limb, but they were slow, especially dorsi-flexion at the ankle. Both knee-jerks were exaggerated, ankle clonus was obtained and the plantar reflex gave an extensor response on both sides.

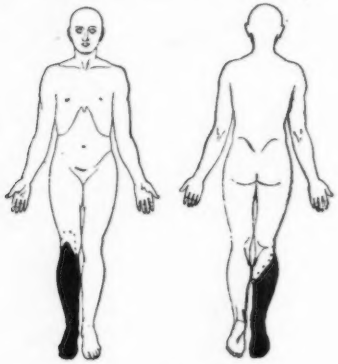


FIG. 4.

To show the loss of sensation in Case 17, due to an injury to the spinal cord.

The dark area represents the parts insensitive to cutaneous painful stimuli, and also to the pain of excessive pressure; and yet over this area light touch and the tactile element of pressure were appreciated.

The right leg below the knee was insensitive to prick and to all degrees of temperature. But, over the whole of this area, he could appreciate all tactile stimuli and could localise accurately the spot touched or pressed upon. Yet it was not possible to produce pain anywhere over the right leg and foot by excessive pressure, although he fully recognised its gradual increase.

	R.	L.
Outer aspect of leg	No pain at 16 ..	Pain at 5.
Inner aspect of leg over tibia	No pain at 10 ..	Pain at 4.
Front of thigh above the limits of the analgesia. ..	Pain at 5	Pain at 5.

Thus, in the former case, where the analgesia over the leg was produced by division of nerve-trunks, a large area of skin was sensitive to the pain of deep pressure, although insensitive to all painful cutaneous stimuli. Only over the foot, where deep touch evoked no response, was the pain of pressure abolished.

But, where the loss of sensation was produced by a lesion of the spinal cord (Case 17), the pain of pressure was absent over parts insensitive to prick, in spite of the complete integrity of the sensations of deep touch, and of the power of recognising the gradually-increasing pressure.

These two patients were chosen as examples, because the site of the loss of sensation was so nearly alike in the two cases. An equally striking contrast is given by

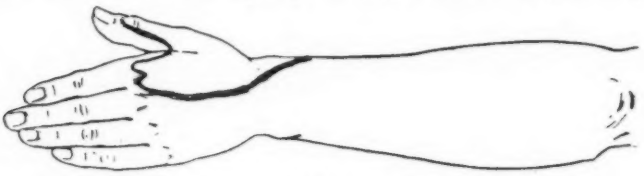


FIG. 5.

To show the loss of cutaneous sensibility on the dorsum of the hand produced by experimental division of the radial (ramus superficialis nervi radialis) and external cutaneous nerves in the neighbourhood of the elbow.

comparing the sensory condition of a part to which all the cutaneous nerves have been divided, with the loss of sensation produced by a pure lesion of the spinal cord.

On April 25, 1903, the radial and external cutaneous nerves were divided at the elbow in one of us. The area on the hand, which became entirely insensitive to all cutaneous stimuli, including the pain of a prick and of the interrupted current, is shown on Fig. 5. But pressure was appreciated and, when increased, caused pain even more readily than over a similar part of the sound hand. The readings given by the algometer were as follows :—

	L. (affected).	R. (sound).
Over the dorsum of the hand	3·5	3·5
In first interosseous space	2·0	3·5
	2·0	4·0

Compare with this dissociated sensibility to painful stimuli, the sensory condition of the hands in Case 13, a patient suffering from syringomyelia. The whole of the area marked on Fig. 6 was insensitive to prick and to the painful interrupted current. Tactile sensibility was everywhere perfect; he could localise accurately the spot touched, or pressed upon, and appreciate passive movements of all the joints. But pain could not be produced by pressure anywhere within the area of cutaneous anal-

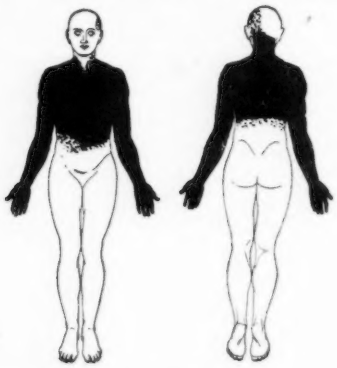


FIG. 6.

To show the loss of sensation to cutaneous painful stimuli in a case of syringomyelia (Case 13). The whole of the area marked black was insensitive to the pain of excessive pressure.

gesia, although he recognised the gradually increasing stimulation.

	R.	L.
Palm of hand	No pain at 15	No pain at 15.
Thenar eminence	No pain at 15	No pain at 15.
Sole of foot	Pain at 4	Pain at 4.

Thus, in conclusion, we have found that, when sensibility to pain is abolished in consequence of an intramedullary lesion, all forms of painful stimuli are simultaneously affected. But, if the lesion be situated in the peripheral nerves or posterior roots, the painful impulses produced by excessive pressure are not interrupted, unless deep sensibility is abolished as a whole.

Section 2.—Heat and Cold.

Great as is the contrast between the grouping of painful afferent impulses in the peripheral and central nervous system, those for sensations of heat and cold undergo an even more complete redistribution. Destruction of either the epicritic or the protopathic fibres interferes with the afferent impulses, both for heat and for cold. If epicritic sensibility is abolished, the patient cannot discriminate intermediate degrees of heat and cold, but remains sensitive to the more extreme temperature stimuli. After a peripheral nerve has been divided, the protopathic system recovers first; this recovery endows the part with sensibility to cold below 20° C. and heat above about 40° C.

Injury to peripheral nerves or posterior roots abolishes, to some degree, sensibility both to heat and cold; recovery restores to the part sensibility to both forms of thermal stimulation.

But, when the lesion is situated within the spinal cord, areas may be found insensitive to heat but sensitive to cold, or *vice versâ*. An intramedullary lesion may even destroy all sensibility to one form of thermal stimulation, leaving the parts completely sensitive to the other. Evidently the whole arrangement of thermal impulses has undergone a change within the spinal cord. All impulses which subserve sensations of heat, whether they arrive by way of the epicritic or protopathic systems, become a single intramedullary group. In a similar manner all thermal impulses, whatever their source, which can produce sensations of cold, are brought together within the spinal cord.

G. G. A. (Case 8) was a good example of this condition. Sensation was disturbed down the right half of the trunk and over the right leg, whilst the left leg was stiff and spastic. The right knee-jerk was brisk, but the left was exaggerated, and clonus was obtained from the left ankle. He was analgesic below the level of the nipple on the right side (Fig. 7). Cold was appreciated everywhere above the right knee; but the outer aspect of the right leg and the whole of the right foot were insensitive to stimulation by cold in all degrees (Fig. 8). Heat from 35° C. to 65° C.

was everywhere appreciated correctly, except that, over the parts insensitive to prick, he did not experience the usual sensation of burning pain when the temperature rose above 50° C.; and yet he could clearly appreciate the increasing heat when the stimulus changed from 50° C. to 60° C.

Here no cold stimulus produced a sensation over parts of the leg, where all temperatures from 30° to 60° C. caused a sensation of heat. Such a total dissociation of the sensations of heat and cold never occurs from division of peripheral nerves.

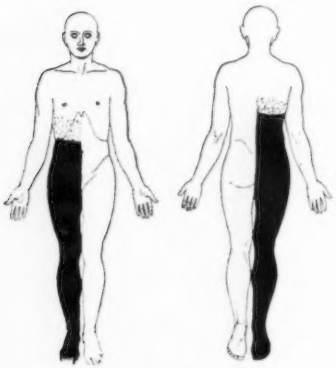


FIG. 7.

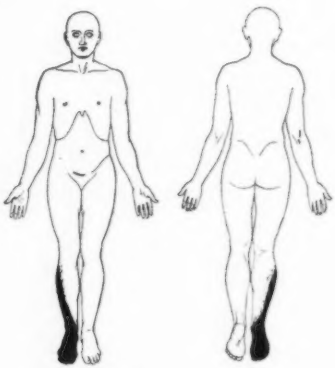


FIG. 8.

Both figures show the loss of sensation produced in Case 8, by disease of the cervical region of the spinal cord. Fig. 7 shows the extent of the loss to prick. Fig. 8 that of the loss to all degrees of cold. Sensation was everywhere present to all degrees of heat.

A similar dissociation of sensibility to heat and cold, but in the reverse direction, was seen in Cyril W. F. (Case 7). In consequence of an injury to the neck, due to diving into shallow water, this young man produced weakness of the right arm, accompanied by loss of sensibility to pain, heat and cold over the left half of the body, each area of loss of sensation having a different extent (Figs. 9 and 10). From the level of the ensiform cartilage downwards to the penis, he was insensitive to all degrees of heat only; temperatures between 30° and 60° caused no sensation of warmth. But,

over this area, he was sensitive to all cold stimuli, recognising them at once.

Here, again, the sensibility to heat and to cold were dissociated. Moreover, those parts insensitive to heat were insensitive to all degrees from 30° C. to 60° C., a condition which never occurs from any lesion of peripheral nerves.

In the case of Mrs. G. (No. 3), the loss of sensation to various thermal stimuli was tested with extreme care on many occasions, and she was found to be insensitive on the left leg to all degrees of temperature between 0° C. and 60° C.

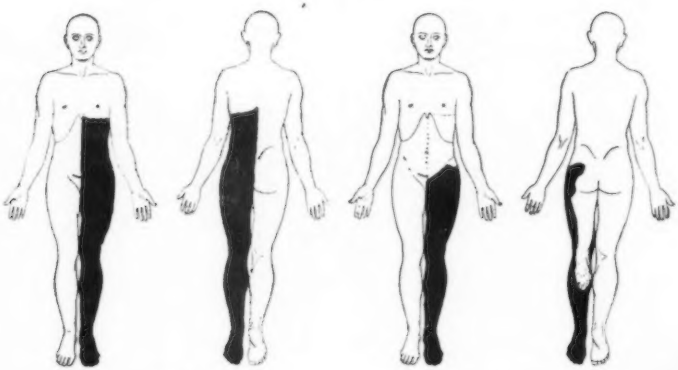


FIG. 9. FIG. 10.

To show the loss of sensation to heat (fig. 9) and to cold (fig. 10) due to injury to the cervical spinal cord (Case 7).

As she was also insensitive to pain over the same parts, it might have been urged that in this case both protopathic and epicritic sensibility had been abolished. The falsity of such a supposition is at once evident from the fact that she was everywhere sensitive to light touch, and that the compass test gave normal results over the affected areas. This condition can never occur from a lesion of peripheral nerves or posterior roots.

An exactly similar loss of sensation to all grades of heat and cold appeared in Case 2, in consequence of an injury to the cervical spine. This completely abolished sensibility to

pain, heat and cold over the left leg and left half of the body, leaving all forms of tactile sensibility entirely unaffected.

In conclusion, we find that the afferent impulses produced by thermal stimuli are differently grouped in the peripheral nerves and in the spinal cord.

(1) Sensibility to heat may be abolished without coincident disturbance of that to cold, and *vice versâ*.

(2) When sensibility to heat is disturbed in consequence of an intramedullary lesion, the patient no longer appreciates any thermal stimulus between 30° C. and 60° C. That is to say, insensibility is absolute to both intermediate and extreme degrees.

(3) Insensibility to heat and cold may be absolute, and yet the patient may be able to recognise the lightest tactile stimulation and to discriminate the two points of the compasses. Such conditions can never occur from a lesion of peripheral nerves only.

Section 3.—*Light Touch and Deep Touch.*

One of the most characteristic features of the loss of sensation following division of peripheral nerves, is the frequency with which light touches are not appreciated, although deep touch and pressure are immediately recognised.

But, when the lesion is purely intramedullary, all these sensory impulses come and go together. Thus, in a characteristic instance of Brown-Séquard paralysis (No. 1), the patient was unable to recognise either light, touch or pressure, applied within the affected area on the right half of the body. The whole of the right leg was insensitive to stimulation with cotton-wool. No reaction could be obtained to any of von Frey's hairs, although No. 5 (*cf.* p. 542) was promptly recognised over the left leg. Pressure, even of many kilograms, produced no sensation, unless the position of the muscles was shifted. Yet this patient recognised accurately any alteration in position, or any movement produced passively in the insensitive leg. Such a condition cannot possibly arise from a lesion of peripheral nerves.

Thus, whenever sensibility to touch is abolished on the opposite half of the body, in consequence of an intramedullary lesion, we believe that all forms of tactile stimuli will be found to be affected. The peripheral afferent impulses for touch and pressure, arriving by way of the epicritic and deep systems, become combined in the spinal cord, so that the lightest perceptible touch produces a minimal tactile impulse, which passes up the spinal system among the tactile impulses evoked by the severer stimulus of pressure.[1]

Section 4.—Passive Position and Movement.

After division of peripheral nerves, the recognition of passive movement, and of the position into which any part of the limb has been placed passively, is strikingly associated with the integrity of that system of nerves concerned with the maintenance of deep sensibility. So long as only the cutaneous afferent nerves are divided, the patient can appreciate all movements of the joints, is sensitive to pressure, and complains of pain when this pressure is increased. There is a close association in the peripheral nervous system between sensibility to pressure, the pain of excessive pressure and the power of recognising passive movement and position. All three forms of sensibility are present or absent together after lesions of the peripheral nerves, and all three depend upon the integrity of those afferent fibres which run with muscular nerves.

With an intramedullary lesion, on the other hand, all recognition of the position assumed passively by a limb is entirely dissociated from the afferent impulses produced by pressure. Both groups are equally dissociated from those impulses which underlie the recognition of the painful nature of excessive pressure.

In fact, Case 1 shows that the patient may be able to appreciate passive position and movement, although totally insensitive to every other sensory stimulus. This man was suddenly seized with loss of power in the left hand and leg, accompanied by absolute loss of sensibility to pain, heat

[1] For a further consideration of the relation between sensations of light and deep touch, *cf.* p. 575.

and cold over the opposite half of the body. His power of movement was gradually restored, but the loss of sensation remained. Insensibility to touch and pressure accompanied the analgesia and thermo-anæsthesia, and excessive pressure produced no pain. But, in spite of this profound loss of sensation, the sense of passive position and movement remained undisturbed in both lower extremities. The most careful testing failed to show a difference between any parts of the two legs in the power of recognising movement or position passively assumed. Here, in spite of the absence of all sensibility to deep touch and pressure, the sense of passive position was not disturbed.

In the case of James Y. (No. 10) an exactly converse condition was present; sensibility to touch and pressure was present, but he was unable to recognise the position into which his limbs had been placed, and failed to perceive even the most extensive movement. In consequence of a metastatic growth within the spinal cord, this man had rapidly lost power in both legs. When seen by us a month after the onset, both legs were flaccid and paralysed, and sensibility to painful and thermal stimuli was abolished below the knees and diminished over the area shown in Figs. 11 and 12; light touch and pressure could be recognised everywhere. But he was unable to tell the position into which his legs had been placed; he could not recognise even the grossest movements at the hip, the knee or the ankle. With this loss of appreciation of passive position and movement was associated a complete absence of discrimination of two points, even when separated to a distance of 20 cm.

This dissociation of the sense of passive position from the impulses of all other forms of sensation, except the discrimination of two points, is peculiarly well illustrated by the case of Charles B. (No. 2). At the time he was first seen, motion was impaired in the left leg, and sensibility to pain and temperature abolished in the right leg and right half of the body. He could appreciate passive movement and the position of all his limbs perfectly. But, in conse-

quence of the operation for the removal of the fractured portion of the spine, he lost the power of recognising passive movement and position of the left leg, *i.e.*, the leg in which all other sensory functions were perfect.

Such loss of the sense of passive movement and position on the opposite side of the body to the analgesia and thermo-anæsthesia is the rule in Brown-Séquard paralysis, and is illustrated by several of our cases.

In conclusion, we have been able to show that, in consequence of an intramedullary lesion, every other form of

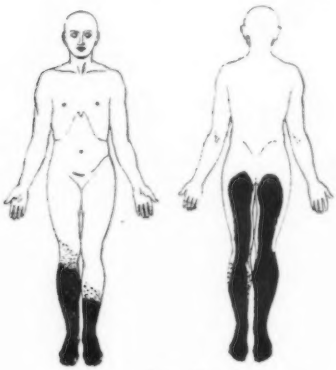


FIG. 11.

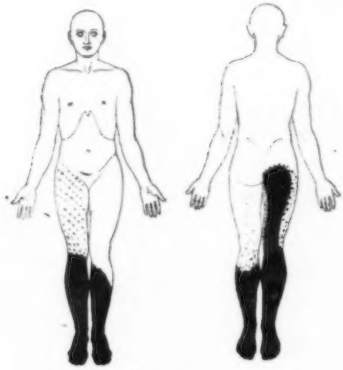


FIG. 12.

To show the loss of sensation produced by a metastatic growth within the spinal cord in Case 10.

Fig. 11. Shows the extent of the loss to pain.

Fig. 12. Shows the extent of the loss to heat and cold.

The extent of the loss of sensation to painful and thermal stimuli varied considerably from day to day, and the areas of total loss were bounded in every case by parts of diminished sensibility.

sensibility may be abolished in a part which still remains sensitive to passive movement. (Case 1.)

Secondly, we have shown that the patient may be unable to recognise even the grossest passive movements, and be entirely unaware of the position of his legs, although they are sensitive to all tactile and pressure stimuli. (Case 10.)

Thirdly, we can bring forward a number of instances where the sense of passive position and movement was dis-

turbed in one leg, whilst every other loss of sensation was to be found in the leg of the opposite side. (Case 2.)

Such dissociation of the impulses underlying the recognition of passive position and movement from the impulses of the other forms of deep sensibility cannot occur in consequence of injury to peripheral nerves.

Section 5.—The Compass-Test.

One of the most remarkable differences between the loss of sensation produced by division of a peripheral nerve and that due to a lesion within the spinal cord is revealed by the compass-test. When a nerve has been injured, sensibility to light touch is the first to be disturbed; with this failure to appreciate careful stimulation with cotton-wool over hairless parts, the compass-test will be invariably found to give results considerably below the normal accuracy. In fact, from the point of view of the peripheral nervous system, the compass-test is an accurate measure of tactile sensibility of the skin. For this purpose Head and Sherren [13] found it most useful, when carried out by the modified method described in their paper.

But, when the lesion is situated within the spinal cord, sensibility to light touch and the power of discriminating two points may be strangely dissociated.

In order to demonstrate this characteristic dissociation, all forms of tactile sensibility must be preserved. For it is obvious that, if the part to be tested is insensitive to touch and pressure, the compass-test will fail from want of afferent impulses. A diminution of tactile sensibility will produce a less accurate compass record, not because the special power of discriminating two points is lessened, but on account of the diminished acuity of the tactile impressions that are to be discriminated. [Case 4, p. 666.]

We have, therefore, selected those cases only where all forms of tactile sensibility were perfect, and yet the power of discriminating two points was gravely disturbed.

In Case 10, a man, aged 61 years, had become rapidly paralysed in both legs in consequence of a growth within the

spinal cord [Figs. 11 and 12.] With this paralysis, he had lost the power of appreciating the position of his limbs to an unusual degree, and was unable to discriminate the two points of the compasses when widely separated. Over the external surface of the leg, he failed entirely at 15 cm. and at 20 cm. over the front of the thigh ; over the palm of the left hand, a part unaffected by paralysis of motion or sensation, he gave correct answers at a distance of 1·5 cm. At this time, there was no part over which he did not at once respond to light touches, or to pressure, and this grossly defective power of discriminating two points cannot, therefore, have been due to altered tactile sensibility.

This dissociation is shown in a peculiarly striking manner by Case 16. The patient, a man, aged 32 years, had suffered for four years from symptoms of a growth within the cervical portion of the spinal cord. This produced a loss of sensation over the arms and trunk shown on Figs. 13 and 14 ; but both legs remained sensitive to all painful, thermal and tactile stimuli. With the finest tests, no difference could be discovered in the tactile sensibility of the two legs. With von Frey's hairs, No. 5 (*cf.* p. 542) could be appreciated everywhere over the soles of both feet ; No. 4 produced no sensation on either limb. And yet the power of discriminating two points was gravely diminished over the left foot and leg. Over the sole of this foot, he answered badly when the points were separated to a distance of 8 cm.

$$8 \text{ cm. } \frac{1 \mid 10 \text{ R.}}{2 \mid 5 \text{ W. } 5 \text{ R.}}$$

whereas, over the right sole, his answers were perfect at 4 cm.

$$4 \text{ cm. } \frac{1 \mid 10 \text{ R.}}{2 \mid 10 \text{ R.}}$$

Over the outer aspect of the left leg, he failed with the points separated to 15 cm.

$$15 \text{ cm. } \frac{1 \mid 4 \text{ W. } 6 \text{ R.}}{2 \mid 5 \text{ W. } 5 \text{ R.}}$$

but over a similar part of the right leg, his answers were uniformly correct at 8 cm.

These two cases show that, within the spinal cord, the impulses which underlie the power of discriminating two points are separated from those of tactile sensibility. Having passed up in the epicritic system, they become dissociated in the spinal cord from the remaining forms of epicritic sensibility. Within the spinal cord, they pass on in close association with the impulses which underlie the recognition of passive movement and position of the limbs, impulses which have reached the cord by means of the system of fibres subserving deep sensibility.

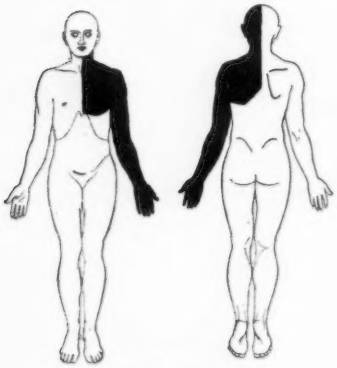


FIG. 13.

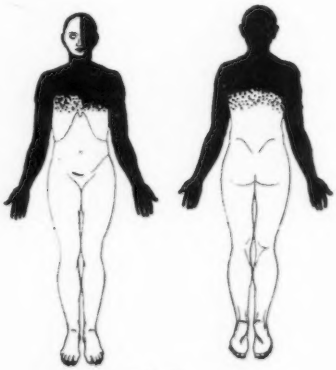


FIG. 14.

To show the loss of sensation in a case of disease of the medulla oblongata and cervical portion of the spinal cord [Case 16.]

Fig. 13. Shows the loss of sensation to painful stimuli. Fig. 14, the loss to heat. To cold, the anæsthesia was slightly less extensive [*vide* p. 731].

The only loss of sensation in the lower extremities consisted of profound loss of the sense of passive position and movement, and of defective sensibility to the compass-test in the left leg.

CHAPTER III.

The Inter-Relation of Afferent Impulses in their Passage up the Spinal Cord.

In the previous chapter, we have shown that afferent impulses, which reach the central nervous system by way of the peripheral nerves, undergo rearrangement within the spinal cord. We shall now attempt to trace their course

upwards, indicating as far as possible the relation of each set of impulses to the others.

More than fifty years ago, Brown-Séquard stated that division of one half of the spinal cord caused loss of sensibility to pain on the side opposed to the lesion, and that motion was affected on the same side.

Led by the results of his experiments on animals, he examined more carefully the condition of sensation in patients suffering from what was called spinal hemiplegia. He attempted to show that this condition was the result of disease or of injury to one half of the spinal cord, which produced paralysis of voluntary movement on the one side, and anæsthesia on the other side of the body.

"Experiments on animals and a great many clinical facts," he says [7], "show that an injury, destroying a small zone, or dividing the whole lateral half of the spinal cord in the cervical region, produces the following symptoms:—

"1st.—Paralysis of the voluntary motor conductors on the same side.

"2nd.—A paralysis also of the vasomotor conductors on the same side, and, as a consequence, a greater afflux of blood and a higher temperature. . . . . .

"5th.—There is anæsthesia of all kinds of sensibility, excepting the muscular sense, in the side opposite to that of the lesion in the spinal cord, owing to the fact that the conductors of sensitive impressions from the trunk and limbs decussate in the spinal cord, so that an injury in the cervical region of that organ in the *right* side, for instance, alters or destroys the conductors from the *left* side of the body.

"6th.—There is some degree of anæsthesia also on the side of the lesion, in a very limited zone, above the hyperæsthetic parts, and indicating the level of the lesion in the cord. This anæsthesia is due to the fact that the conductors of sensitive impressions reaching the cord through the posterior roots, at the level or a little below the seat of the alteration, have to pass through the altered part to reach the other side of the cord."

This admirable summary is followed by the narration of

two cases, observed by Brown-Séquard himself, with a fulness rarely equalled in the present day [7].

His results have been attacked from two points of view. It was pointed out that the lesions produced by disease were crude and ill defined, and commonly affected both halves of the spinal cord. Moreover, *post-mortem* examinations on those who had suffered from Brown-Séquard paralysis are uncommon, except in cases complicated by grave general disease, which renders difficult an accurate investigation of sensibility.

Physiologists attempted to solve the question by means of animal experiments, carried out with all modern surgical precautions. Some (especially Turner [37]) supported the original view of Brown-Séquard, others (Schiff, Mott [19], Rothmann [24], Schuster [26]), concluded that afferent impulses did not cross in the diagrammatic manner he described. Moreover, many experimenters have shown that hemisection in the dorsal region produces ascending degeneration in both the same and the opposite half of the spinal cord. It is therefore certain that to afferent impulses, arriving by way of the posterior roots, both crossed and uncrossed paths are open.

But of the nature of the afferent impulses passing by these paths, experiments on animals can tell us little. It is difficult to conclude whether they are non-sensory afferent impulses, concerned with what may be called the higher reflexes, or whether they are ultimately concerned with some form of sensation. For every examination of sensation is in reality a psycho-physical experiment, where the extent of the mental error can entirely obscure the physical result. The nature and extent of these errors we can estimate only in man. Moreover, it is not sufficient to note the response to a stimulus; we must hear from the lips of the patient his experiences under stimulation. Supposing he starts and withdraws his foot when pricked, we cannot conclude that he experiences pain. It may be that his withdrawal is the reflex response to pressure with a pointed object. He alone can tell us the nature of his sensations. (Compare p. 644.)

Thus, we are compelled to fall back upon the results of disease. But the destruction produced by disease is greatly wanting in precision compared with an experimental lesion. Moreover, on account of lack of time, or forethought, or the progressive nature of the disease, many clinical records are wanting in necessary fulness and precision. Thus, on the one hand, are those who, for the sake of the definiteness of the lesion, cling to the results of animal experiment, which can demonstrably lead to elementary conclusions only concerning sensation; on the other stand the clinicians, with their imperfect records and ill-defined terms.

We do not attempt to base our conclusions on the verification of the position of the lesion by *post-mortem* examination. For, in most cases where a microscopical examination was possible, the general condition of the patient had been unfavourable to that rigid and repeated examination by which alone the condition of sensibility can be established.[1] We shall assume that an intramedullary lesion disturbs the motor functions, and alters the reflexes on the same side of the body. This no one has doubted. Then we shall examine the effect produced by an intramedullary lesion upon the patient's answers to the series of sensory tests detailed in Chapter I. From these we shall deduce the manner in which the sensory impulses are grouped in their passage up the spinal cord, and correlate the loss of sensation with the motor disturbance.

Such study of the nature and relation of sensory impulses, when disturbed by an intramedullary lesion, has led to more fruitful results than the anatomical method, which demands in every instance that the lesion should be demonstrated to be unilateral. For a complete unilateral lesion, especially if it be the result of progressive disease, will certainly cause functional and even microscopical changes in the opposite half of the cord.

We have arranged on Table 1 (p. 570), eight cases of Brown-Séquard paralysis observed by ourselves. In every instance the motor disturbance was limited to the one half

[1] We have rejected two cases in which a complete microscopical examination was made on account of faulty sensory records.

Case.	Disease.	Motor Disturbance.	Sensory		
			At Level of Lesion.	Remote (Due to Interference	
				Light Touch.	Pressure Touch.
No. 1. F. C. Appendix, p. 646.	Probably hæmorrhage into the spinal cord. Sudden onset, Dec. 13, 1903	At first weakness of L. arm. No spasticity	None ..	R. leg and R. half of body below level of umbilicus	R. leg and R. half of body below level of umbilicus
No. 4. A. H. Appendix, p. 662.	Fracture of fourth cervical vertebra in 1901	Spasticity and weakness L. leg and L. arm	None ..	Slightly diminished over R. foot (p. 666).	Slightly diminished over R. foot (p. 666).
No. 2. C. B. Appendix, p. 652.	Fracture of third cervical vertebra in 1904	*Before operation*— Weakness of R. arm and leg	None ..	None ..	None ..
	Operation June 25, 1905	*After operation*— Increased weakness of R. arm and leg	None ..	None ..	None ..
No. 3. G. G. Appendix, p. 656.	Probably hæmorrhage into the spinal cord. Sudden onset, Feb. 18, 1898	Spasticity and weakness R. leg	None ..	None ..	None ..
No. 5. W. M. Appendix, p. 667.	Fracture of cervical spine in 1904 (Radiograph shows disintegration of sixth and seventh cervical vertebra)	*Local*— R. hand, wasting of small muscles *Remote*— R. arm and R. leg, spasticity	None ..	None ..	None ..
No. 6. J. M. Appendix, p. 672.	Injury to cervical spine from diving into shallow water in 1886	*Local*— L. hand, wasting of small muscles *Remote*— Spasticity of L. leg	None ..	None ..	None ..
No. 7. C. W. F. Appendix, p. 677.	Injury to cervical spine from diving into shallow water in 1905 (Radiograph shows changes in body of sixth cervical vertebra)	*Local*— R. hand, slight wasting small muscles *Remote*— None	None ..	None ..	None ..
No. 8. G. G. A. Appendix, p. 681.	Disease of the cervical cord, possibly syphilitic. Onset 1904. Seen by us 1906	*Local*— L. hand, slight wasting small muscles *Remote*— Spasticity L. leg	None ..	None ..	None ..

Disturbance.					Reflexes.
(with Long Sensory Paths).					
Pain.	Cold.	Heat.	Passive Position and Movement.	Compasses.	
R. leg and R. half of body below level of umbilicus	R. leg and R. half of body below level of umbilicus	R. leg and R. half of body below level of umbilicus	No loss ..	L. no loss, R. no response.	Knee-jerks equal. No ankle clonus. Plantars flexor.
R. leg and R. half of body	R. leg and R. half of body	R. leg and R. half of body	No loss ..	No loss ..	L. knee-jerk + +. R. knee-jerk normal. L. ankle clonus. R. no ankle clonus. L. plantar extensor. R. plantar flexor.
L. half of body and greater part of L. arm	L. half of body and greater part of L. arm	L. half of body and greater part o L. arm	No loss ..	No loss ..	R. knee-jerk + +. L. knee-jerk normal. R. ankle clonus. L. no ankle clonus.
Same ..	Same ..	Same ..	Profound loss. R. arm and R. leg	Not tested ..	R. plantar extensor. L. plantar flexor.
L. lower extremity up to groin	L. lower extremity up to groin	L. lower extremity up to groin	R. foot ..	R. foot ..	R. knee-jerk + +. L. knee-jerk normal. R. ankle clonus. L. no ankle clonus. R. plantar extensor. L. plantar flexor.
L. leg, L. half of body and strip on L. arm	L. leg, L. half of body and strip on L. arm	L. leg, L. half of body, and strip on L. arm	No loss ..	No loss ..	R. knee-jerk greater than L. R. ankle clonus. L. no ankle clonus. R. plantar extensor. L. plantar flexor.
R. half of body below umbilicus, and R. leg, except over sacral areas	R. half of body below umbilicus, and R. leg, except over all sacral areas	R. half of body below umbilicus, and R. leg, except over all sacral areas	No loss ..	No loss ..	L. knee-jerk + +. R. knee-jerk normal. L. ankle clonus. R. no ankle clonus. L. plantar extensor. R. plantar flexor.
Front of L. leg and thigh. All sacral areas unaffected	L. leg, except all sacral areas	L. leg and L. half of body to level of ensiform and angle of scapula	No loss ..	No loss ..	R. knee-jerk greater than L. No ankle clonus. R. plantar doubtful. L. plantar flexor.
R. leg and R. half of body to level of ensiform	R. foot and outer aspect. R. leg below knee	No loss ..	No loss ..	No loss ..	Left knee-jerk + +. R. knee-jerk normal. L. ankle clonus. R. no ankle clonus. L. plantar extensor. R. plantar flexor.

of the body, and in no case was the same form of sensation disturbed on the two sides.

In this chapter we shall deal with the disturbances of sensation remote from the level of the lesion. As Brown-Séquard pointed out, a lesion of the spinal cord not uncommonly produces some degree of anæsthesia on the side of the lesion, due to an interruption of sensory impulses at the level of the disease. These local manifestations will be reserved for Chapter IV., while in the present chapter we shall deal with the disturbances of sensation in parts remote from the level of the lesion.

Section 1.—*Pain, Heat and Cold.*

The close relation between the impulses for sensations of pain and temperature has long been universally recognised. Easily investigated and clearly defined, the loss of sensibility to pain, heat and cold is well described in most cases of disease of the spinal cord, however insufficient may be the remainder of the record. Petrén [21] has gathered together records of 137 unilateral lesions of the spinal cord in a monograph on this subject. He concludes that, in 128 cases where the condition was adequately recorded, sensibility to pain, heat and cold was abolished on the side opposite to the lesion.

Thus, whatever may happen to the impulses which underlie other forms of sensibility, there can be little doubt that, in man, those for pain, heat and cold cross to pass up the opposite side of the spinal cord.

To these cases may be added the eight instances of Brown-Séquard paralysis in the Table on p. 570. In all it will be found that the motor disturbance was strictly unilateral, and the remote loss of sensation was confined to one side; in all, sensibility to pain, heat and cold was disturbed on the side opposed to that of motion.

We have already described the character of this loss of sensation in Chap. II. When sensibility is abolished to pain, from an intramedullary lesion, no normally painful stimulus, whether it be a prick, the interrupted current, or excessive pressure, will produce a painful afferent

impulse. In the same way, sensation is lost to all degrees of heat or of cold, except at the edges of an ill-defined area of thermo-anæsthesia. Here the more extreme degrees of heat or of cold may produce a sensation, because they are a more intense thermal stimulus than the intermediate degrees (*cf.* Piltz [22]); but, if the insensitive area has well-defined borders, all forms of the specific sensibility to heat or to cold are abolished altogether.

Although the impulses for pain, heat and cold are so closely associated in their passage up the spinal cord on the side opposed to their point of entry, they do not travel inextricably intermingled. Not only can the three forms of sensibility be destroyed over different skin areas, but it is possible that one may even escape entirely.

In Case 8, it is almost certain that at one time sensations of pain, heat and cold were lost over the right half of the trunk and over the right leg. But, when the patient was first seen, the dissociation was pronounced. He could not appreciate a prick, or the pain of excessive heat, or the painful faradic current, over the right half of the body below the costal margin. But sensibility to all degrees of cold was lost over the right leg below the knee only, whilst temperatures between 35° C. and 65° C. could be everywhere recognised. (Figs. 7 and 8.)

It is particularly in lesions of the cervical portion of the spinal cord that the three forms of sensation tend to be unequally affected. In August, 1905, C. W. F. (Case 7) injured his neck from diving into shallow water; when seen by us in November, the loss of sensation was more extensive to heat than to cold, to cold than to pain (Figs. 15, 16, 17). Moreover, this patient showed another phenomenon not uncommon in lesions of the upper part of the cord; the analgesia occupied the front of the thigh and leg, but did not extend on to the sacral areas. To a less degree, this was the case also with the loss of sensation to cold. Thus, a lesion in the cervical region abolished sensation over the opposite leg, without interfering with the impulses from the terminal portions of the spinal cord. This was extremely well shown in the case of J. M. (No. 6), who twenty years before we

first saw him, had injured his cervical spine by diving into shallow water. In this instance, the extent of the loss to pain was greater than that to cold and to heat.

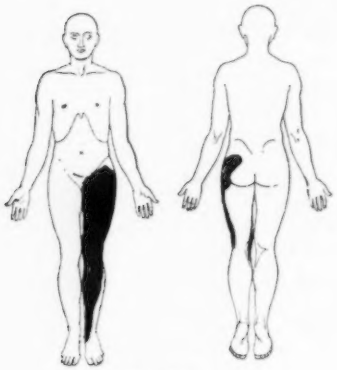


FIG 15.

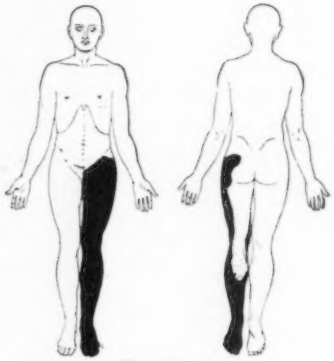


FIG. 16.

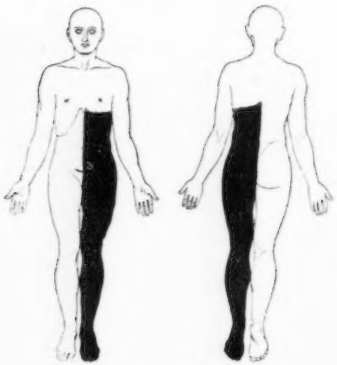


FIG. 17.

To show the loss of sensation in Case 7.
Fig. 15 shows the extent of the loss of sensation to painful stimuli.
Fig. 16 shows the loss to cold.
Fig. 17 shows the loss to heat.

When the loss of sensation produced by an intramedullary lesion occupies the opposite half of the body only, the

boundary between parts of normal and abnormal sensibility is usually ill defined. This might have been foretold from our knowledge of the irregular distribution of such lesions.

But, occasionally, the upper border of the loss of sensation to pain, to heat and to cold can be determined with unusual certainty. Such definiteness of outline must signify that all impulses passing upwards have been interrupted completely at a certain level.

Now it would seem from some of our cases that, even when the impulses for pain, heat and cold are interrupted after they have crossed the cord, the situation and shape of the border between parts of normal and abnormal sensibility point to a segmental arrangement. (For a full consideration of intramedullary segmentation *vide* Chap. V.).

In conclusion, we believe :—

(1) That afferent impulses for pain, heat and cold cross the spinal cord to pass up the opposite side.

(2) That the remote loss of sensation may affect to a varying degree the impulses for the three forms of sensibility.

(3) The borders of this remote loss of sensation on the opposite side of the body to the intramedullary lesion usually merge gradually into parts of normal sensibility. But, occasionally, they correspond to the borders of intramedullary segments.

Section 2.—*Touch and Pressure.*

In a previous chapter, we showed that the disturbance of tactile sensibility, due to an intramedullary lesion, differed fundamentally from that produced by injury to a peripheral nerve, or posterior nerve root. For, when a peripheral nerve has been divided, a considerable extent of the anæsthetic area remains sensitive to pressure, but insensitive to light touch. But, if the lesion lies within the spinal cord, tactile sensibility is diminished, or abolished as a whole, whether the touch be caused by the lightest movement of hairs, by stimulation with cotton-wool, or by pressure. Occasionally, it is true, pressure produces the sensation of touch, when

the less insistent forms of tactile stimulation fail to be appreciated. If an intramedullary lesion has destroyed the impulses associated with the sensation of light touch, it will also interfere with those that underlie sensibility to pressure; that pressure can still be appreciated is due solely to the fact that it is a greater tactile stimulus, not to any difference in path between the impulses for light touch and for pressure.

In the previous section, we showed that the impulses for pain, heat and cold crossed the spinal cord to ascend on the opposite side. Here we shall consider the intramedullary course of tactile impulses.

The clinical records of cases of Brown-Séquard paralysis are more especially defective with regard to the condition of tactile sensibility; most of the stimuli that have been employed for touch are, in reality, tests for the recognition of pressure. Fortunately, however, the impulses for light touch and pressure are combined into one tactile group during their passage up the spinal cord; pressure is only an extreme form of touch. We may, therefore, assume that if, in any case of Brown-Séquard paralysis, it is recorded that pressure was not appreciated, sensibility to light touch was also abolished.

Sensations of touch and pressure are much less frequently disturbed in cases of Brown-Séquard paralysis than those of pain, heat and cold. In the two cases observed with unusual care by Brown-Séquard himself, tactile sensibility was greatly diminished, or lost, over the same parts that were insensitive to pain, heat and cold, on the opposite side of the body to the disturbance of motion. Among 128 cases collected from the literature by Petrén, the anæsthesia to pressure reached the same development as that to pain, heat and cold in 39 only. In every instance the loss of sensibility to pressure was found on the same side as the analgesia.

This in no way proves that a unilateral lesion of the cord produces loss of tactile sensibility. It demonstrates only that, if this form of sensibility is abolished as the remote effect of an intramedullary lesion, the loss of sensa-

tion will be found on the opposite side of the body to the motor disturbance.

We are able to add an excellent instance of this comparatively rare loss of tactile and pressure sensibility in a case of Brown-Séquard paralysis.

F. C. (Case 1), went to bed perfectly well on December 13, 1903. When he tried to get out of bed he found he "could not use his legs," and fell to the ground. On the afternoon of December 14 he found his left arm was weak. By Easter, 1904, his left arm and his left leg had recovered, and he returned to work. He had noticed little wrong with

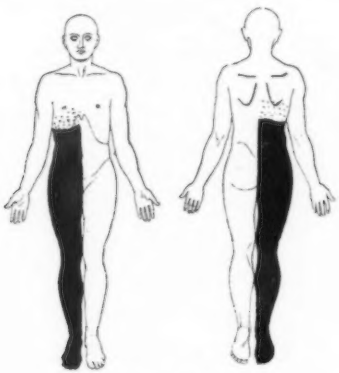


FIG. 18.

To show the loss of sensation to all forms of tactile, painful and thermal stimuli in Case 1. The frontal border was not sharply defined, but merged gradually into parts of normal sensibility.

the other side, but within three weeks of the onset, during his stay in the National Hospital, Queen Square, complete analgesia and thermo-anæsthesia had been discovered over the right half of the trunk and right leg.

He first came under our care in August, 1905, for an attack of acute rheumatism. During this time we discovered the condition of sensation, which has remained substantially unaltered up to the present time.

He now shows the following disturbance of sensation. All sensibility to cotton-wool is abolished over the right leg

and right half of the trunk, as high as the level marked on Fig. 18. Within this area he is totally insensitive to pressure steadily applied; but, if the muscles are rolled from side to side during the act of pressure, he sometimes says, "You are rolling the front of my leg," or, "You are pressing me." This loss of sensation is associated with inability to distinguish the head of a pin from the point, or to appreciate difference in size.

All sensibility to pain is abolished over the same parts, whether the stimulus be a prick, an interrupted current or extreme pressure. Even a pressure of 20 kgms., applied to the outer aspect of the right knee, produces no pain, although, on a similar part of the left leg, the patient complains of pain with a pressure of 4·5 kgms.

The recognition of passive position is perfect in both legs from the toes to the hip.

All loss of motor power has now disappeared; his grasps are equal and there is no stiffness of either leg. The knee-jerks are brisk but equal, and ankle clonus cannot be obtained. On neither side does the plantar reflex give an extensor response.

Thus we conclude, that sensibility to touch and pressure is rarely disturbed in Brown-Séquard paralysis. But, if such a change exists,

(1) It is found on the opposite side of the body to the motor disturbance. (Case 1 and Case 4.)

(2) The impulses of light touch (cotton-wool, von Frey's hairs), deep touch and the tactile element of pressure are lost together. If the borders of the loss of sensation are ill defined, pressure can be appreciated over parts insensitive to stimulation with cotton-wool, in consequence of the greater intensity of pressure as a tactile stimulus. There is no evidence pointing to a separation of sensibility to light touch from that to the tactile aspect of pressure, such as is found in lesions of peripheral nerves.

Section 3.—*Sense of Passive Position and Movement.*

Throughout this paper, in almost every section, our investigations have borne their tribute to the acumen and

accuracy of Brown-Séquard. He stated that the impulses for the sensations of pain, heat and cold crossed to the opposite side of the cord ; ultimately, the impulses for touch, though more rarely disturbed, followed a similar course. Not only our cases, but the whole of the work of the past, when analysed, supports this view.

Much controversy has arisen regarding the further statement of Brown-Sequard, that appreciation of the position into which a limb has been passively placed is affected on the same side of the body as the disturbance of motion and reflexes. Nothing could be more convincing than a perusal of the beautifully recorded cases that came under his personal observation.

Among our observations, the following case (No. 2) is a striking corroboration of the view, that the impulses which underlie knowledge of the position into which a limb has been placed are interrupted on the same side as the motor disturbance. It is a peculiarly unequivocal instance, because the patient was examined when all sense of passive position was perfect ; yet the right leg was spastic, and sensibility to pain, heat and cold was abolished over the left half of the body and left leg. After the operation upon his spine, these signs remained unaltered, but he lost all power of recognising the position into which his right leg had been placed.

His history was as follows : On April 11, 1904, he was thrown out of a dog-cart on to his head. He became unconscious for a few seconds and, when he recovered his senses, found he was completely paralysed down the right side. From the moment of the accident he has been unable to appreciate pain, heat and cold applied to the left leg and left half of the trunk.

In May 1905, when first seen by one of us, a little more than a year after this accident, he walked with a hemiplegic gait, swinging his right hip, so that the toes of the right foot might clear the ground.

Over the left half of the body and the whole of the left leg below the level of the seventh cervical segment, he was insensitive to all forms of pain, heat and cold. Light touch with cotton wool, deep touch and pressure were recognised

everywhere, and were well localised. The sense of passive position was perfect in all four limbs, and he could recognise the position assumed by parts of the limb under electrical stimulation.

On the right side the knee-jerk was greatly exaggerated, ankle-clonus was easily obtained and the plantar reflex gave an extensor response. The left knee-jerk was diminished and no clonus could be obtained in the left ankle; the plantar reflex on this side gave a flexor response.

Thus at this time the patient showed all the signs of that form of Brown-Séquard paralysis where motion is disturbed on the one side, sensibility to pain, heat and cold on the other, touch and pressure and the sense of passive position being entirely unaffected.

On June 25, 1905, Sir Victor Horsley exposed the laminæ of the cervical vertebræ and found that the third neural arch was fractured. All went well until a fortnight after the operation, when the patient noticed that the right side and right leg seemed stiff and cramped and he thought he had lost power.

But, when he was again seen by one of us, the only further change that could be discovered was a loss in the right arm and leg of the sense of position assumed passively. He could not touch his nose with his right forefinger if the eyes were closed. He could not tell into what position the thumb and fingers of the right hand had been placed, unless the movement was violently performed, and movements of the right wrist were badly appreciated. No movement of the toes of the right foot could be recognised.

At the same time, he was sensitive everywhere to stimulation by light touch and pressure, and the point touched was well localised. Thus, the operation had added loss of sense of passive position and movement on the right side to the loss of sensibility to pain, heat and cold, which previously existed over the left half of the body.

Case 16 also illustrates the loss of the sense of passive position and movement on the same side of the body as the disturbance of reflexes and of motion. R. A. H. suffered from a long-standing intramedullary lesion of the bulb and

cervical region of the spinal cord, which had paralysed the left vocal cord and left half of the palate. The left knee-jerk was exaggerated, clonus was obtained from the left ankle and the left plantar reflex gave an extensor response. On the right side, the reflexes were normal and no ankle clonus could be obtained.

Sensibility to all painful and tactile stimuli was disturbed over the whole of the left fore-quarter, but to heat and cold the loss of sensation occupied both upper extremities.

The legs remained normally sensitive to all forms of tactile, painful, and thermal stimulation, but the sense of passive position was profoundly disturbed in the left lower extremity, *i.e.*, on the side of the alteration in motion and reflexes. Out of twenty movements, ten of which were dorsiflexion and ten plantar extension of the left ankle, he gave eleven false answers—

Dorsiflexion	7 W. 3 R.
Plantar extension	4 W. 6 R.

On the right side he made no mistake under similar conditions—

Dorsiflexion	10 R.
Plantar extension	10 R.

Sense of passive position was even more grossly affected in the toes of the left foot, but his answers were faultless and quick from those of the right foot.

Case 3 is another striking instance of a similar inability to recognise the position assumed passively by the toes; this loss was also found in the limb opposite to that insensitive to pain, heat and cold. This patient has been under observation for eight years, and we have therefore had ample opportunity of confirming our observations.

In March, 1898, she was suddenly seized with paralysis of the right leg accompanied by loss of sensation to pain, to heat and to cold in the whole of the opposite limb. Since that time, her condition has remained materially unchanged.

When walking, she drags the right leg, which she holds stiffly, saying she dare not relax this leg, or she would fall. The whole leg is spastic, and voluntary movement is im-

paired by its rigidity; but, in addition, there is some loss of power, especially in the movements of the right hip. There is no wasting, and the electrical response is everywhere normal.

Light touch and pressure are everywhere appreciated and accurately localised. Repeated examination failed to reveal any difference between her answers on the two sides. But the whole of the left leg and thigh below the level of the eleventh dorsal segment is absolutely insensitive to all forms of pain, heat and cold.

In this leg, every movement produced passively is quickly and easily recognised. She made no mistake when the left great toe was moved twenty times into a flexed or extended position. But on the right side, the side of the muscular rigidity and weakness, she failed repeatedly to recognise the position of the great toe. Here, our method of recording the results shows the difference between the condition of the two feet with remarkable distinctness.

Left Great Toe (Analgesia and Thermoanæsthesia).

Flexion	I.	III.	II.	II.	II.
Extension	II.	II.	I.	III.	II.

Right Great Toe (Motor Loss).

Flexion	II.	X.	IIIX.		III.
Extension	XX.	I.	XI.	XXIXX.	

No difference could be observed between her power of appreciating movements of the ankles, knees or hips. Exactly similar results were obtained as far back as 1898.

Here, again, the loss of the sense of a position assumed passively by part of the limb was found on the side of the motor disturbance, and not on the side insensitive to pain, heat and cold.

Thus, we conclude that, with regard to the impulses which underlie our power of recognising the passive position of a limb, Brown-Séquard's original statement was correct.

(1) The sense of passive position and movement is abolished on the same side as the motor disturbance, provided that the loss of motion is entirely unilateral and no single form of sensation is disturbed on both halves of the body.

(2) The loss of the sense of passive position and movement is independent of the tactile impulses and of those which underlie the power of cutaneous localisation. But, as will be shown in the following sections, it is closely associated in Brown-Séquard paralysis with inability to discriminate two compass-points.

Section 4.—The Relation between the Power of Discriminating Two Points and the other Forms of Sensibility.

In a previous chapter, we cited two cases to show that tactile sensibility might be perfect and yet the patient might be unable to discriminate two points applied simultaneously to the skin. Both of these cases revealed incidentally the close association between the loss of the sense of position assumed passively, and the power of discriminating two points.

Now, we have already ample evidence, that the power of recognising the passively assumed position of any limb tends to be disturbed on the same side as motion in Brown-Séquard paralysis. We should therefore expect that the compass-test would also show a diminished accuracy of discrimination on the same side.

But it must be remembered, that the compass-test depends upon two entirely separate factors. In order that the test can be applied, the patient must be able to recognise that he is being touched. If sensibility to touch and pain is lost, the test is obviously inapplicable, because of the total absence of sensation, and not because of any necessary inability to discriminate two points. A diminution of tactile sensibility alone will affect the results of the compass-test, by decreasing the certainty with which the patient knows when he has been touched. This was the reason Brown-Séquard stated that, in two of his cases, the compass-points were not normally discriminated over the non-paralysed limb. In both of these cases, tactile sensibility was profoundly affected on that half of the body opposite to the motor paralysis. (*Cf.* Case 4, p. 666.)

For the same reason Spearman [31], in his careful report, found that the compass-test gave bad results on both

legs. We venture to suggest that on the right leg, which was analgesic and thermo-anæsthetic, this was mainly due to the grave defect in contact sensibility; on the left leg, which was spastic and paretic, the inability to recognise two points was associated with the loss of sense of position. The probability of this explanation is increased by the remarkable raising of the threshold for the compass-test which occurred on the left leg as the result of muscular fatigue, a condition scarcely noticeable in the right leg, where the cutaneous sensory loss was profound.

Since the compass-test depends upon two factors, which may be separated in disease of the spinal cord, it is necessary to choose cases where one of these factors alone is demonstrably unaffected. It is obvious that a diminution of tactile sensibility will diminish the ease with which two points are discriminated; but we have other less elaborate tests for touch, and the true interest and value of the compass-test lies in the revelation of a loss of power of discriminating two points, although tactile sensibility is perfectly maintained.

Case 3 is an instance of such a condition. Over the left leg, sensibility to pain, heat and cold was abolished, whilst in the right foot the patient was unable to recognise the position into which her toes had been placed. To the finest tests all forms of touch were everywhere perfect.

When the compasses were applied longitudinally to the sole at a distance of 2·5 cm., it was found that she was more accurate in her answers on the left foot (sensory loss) than on the right (motor loss).

Left 2·5 cm. $\begin{array}{l|l} 1 & 9\,\text{R.}\ 1\,\text{W.} \\ 2 & 10\,\text{R.} \end{array}$

Right 2·5 cm. $\begin{array}{l|l} 1 & 8\,\text{R.}\ 2\,\text{W.} \\ 2 & 8\,\text{R.}\ 2\,\text{W.} \end{array}$

For some hours before these tests, she had been resting in bed, and she was therefore tested again after walking about 150 metres without a stick. The result was striking. For whereas at 4 cm. distance the test gave perfect results on the dorsum of the left foot, her answers failed grossly over a similar part of the right foot.

Left 4 cm. $\frac{1 \mid 10\text{ R.}}{2 \mid 10\text{ R.}}$

Right 4 cm. $\frac{1 \mid 6\text{ R. }4\text{ W.}}{2 \mid 6\text{ R. }4\text{ W.}}$

This brings out the considerable raising of the threshold for the compass-test which follows exertion, a fact first pointed out by Spearman [31].

On account of the stationary condition of her symptoms and her great quickness and trustworthiness, this patient forms a good instance of the close connection between delicacy in discriminating two points and a recognition of the position of the limb.

When discussing disturbances of the sense of passive position and movement, we cited the case of R. A. H. (No. 16), where an intramedullary lesion of the bulb and cervical spinal cord had produced locally loss of sensibility in both upper extremities, whilst motion and the reflexes were disturbed on the left side only.

In this patient, the remote loss of sensation consisted of a profound disturbance of the sense of passive movement and position in the left foot and ankle. Together with this loss, it was found that his power of discriminating two points was profoundly affected over the left foot and leg. Thus, over the sole of the left foot his answers were bad when the points were separated to 8 cm.

$$8\text{ cm. } \frac{1 \mid 10\text{ R.}}{2 \mid 4\text{ R. }6\text{ W.}};$$

whilst over the right sole they were perfect at 4 cm.

$$4\text{ cm. } \frac{1 \mid 10\text{ R.}}{2 \mid 10\text{ R.}}$$

Over the outer side of the left leg he answered badly at 15 cm.

$$15\text{ cm. } \frac{1 \mid 6\text{ R. }4\text{ W.}}{2 \mid 5\text{ R. }5\text{ W.}}$$

although his answers were perfect at 8 cm. over a similar part of the right leg

$$8\text{ cm. } \frac{1 \mid 10\text{ R.}}{2 \mid 10\text{ R.}}$$

This difference in the sensibility of the two legs to the compass-test could not have been due to any alteration in tactile sensibility. For stimulation with cotton wool was appreciated equally when applied to both feet and to the outer side of both legs; over both soles, von Frey's hairs produced sensations of equal intensity. Stimulation with No. 8 and No. 5 was well appreciated; but stimulation with No. 4 applied to the soles could not be appreciated over either foot.

In conclusion, we believe that, if all forms of tactile sensibility are perfect in Brown-Séquard paralysis, (1) the power of discriminating two points will be found to be diminished over the side of the motor disturbance; (2) there is a close relation between loss of the sense of passive position and movement and the power of discriminating two points; loss of the one is usually associated with grave disturbance of the other.

Chapter IV.

Local Effects of an Intramedullary Lesion.

So far we have neglected the local effects produced by a lesion of the spinal cord, and have dealt solely with its remote results on sensation. The skilled operator can now produce, with the help of rigid asepsis, an experimental division of one half of the cord, of such precision that the sensory effects at the level of the lesion, on the same side as the section, may be negligible. The sensory consequences of a stab in the back, producing a wound of the spinal cord, may also under favourable conditions be entirely remote. Even those remarkable cases of Brown-Séquard paralysis, arising suddenly and presumably due to spontaneous hæmorrhage, may show no permanent sensory changes at the level of the disease.

But, as soon as interference in the conducting paths is due to a growth or to a large hæmorrhage, such as not uncommonly follows fracture of the spine, the disease manifests itself by local disturbances which will form the theme

of this chapter. The greater the extent of the lesion longitudinally in the spinal cord, the wider will be the area upon the surface of the body affected by these changes; and of all local lesions, the most widely extended is that of syringomyelia. At any rate in its early stages, this disease manifests itself by sensory changes on the same side of the body as the disturbance of motion; its signs and symptoms are essentially the local manifestations of a lesion of vast longitudinal extent.

In some cases of syringomyelia, the central growth reaches such proportions that long afferent paths are destroyed, and parts at a distance from the focus of disease become insensitive; but, in essence, syringomyelia is a local disease, producing sensory changes at the level of the lesion.

Section 1.—*The Local Sensory Effects of an Intramedullary Lesion occur on the Same Side as the Disturbance of Motion.*

Every disturbance of cutaneous sensibility, due to the local effect of disease of the spinal cord, lies on the same side as the muscular wasting and loss of motor power, that is, on the side of the maximum lesion. This forms a fundamental difference between the local and remote effects of intramedullary disease.

This rule is well illustrated by the following case of gliosarcoma of the spinal cord (Case 9). In November, 1904, Joseph F. noticed a slight weakness of the left arm; soon afterwards, his neck became stiff, and he complained of tingling sensations in the fingers of the left hand. The weakness of the left arm gradually increased until February, 1905, when it became completely paralysed. During March and April, he became progressively weaker, and his legs began to give way.

In July, 1905, when admitted to the London Hospital, his left arm lay motionless on the bed by his side, completely paralysed for voluntary movement. The small muscles of the hand and, to a less degree, those of the forearm were wasted, but reacted in the normal manner to both the constant and interrupted currents. The grasp

of the right hand was weak and its small muscles were somewhat wasted; all movements were well performed by the remaining muscles of the limb.

Sensation was profoundly disturbed over the whole of the left upper extremity. All sensibility to pain, to heat and to cold was lost over the areas shown in Figs. 19 and 20, extending as high behind as the occipital crest and occupying the whole of the arm, excepting only a small patch on its inner aspect.

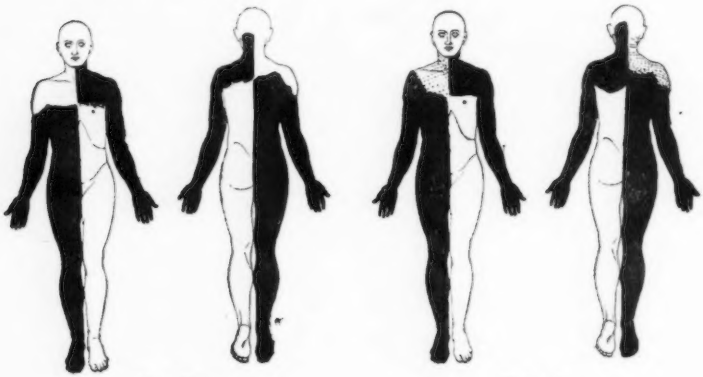


FIG. 19. FIG. 20.

To show the loss of sensation to painful (fig. 19) and thermal (fig. 20) stimuli produced by a glio-sarcoma of the spinal cord (Case 9). In the cervical region the growth destroyed mainly the left half of the cord. The condition in the left upper extremity represented the local, that on the right half of the body the remote effect of the lesion.

Light touches and pressure were everywhere appreciated and well localised, but two points of the compasses could not be discriminated, even at a distance of 5 cm., anywhere over the left hand. Over the right palm, his answers were perfect when they were 2 cm. apart, and he made two mistakes only in twenty applications, when the points were separated by a distance of 1 cm.

All sense of passive position and movement was lost in the left upper limb. When his eyes were closed, he was

unable to indicate the position of his fingers in space or the direction in which they had been moved; and this was also the case with the wrist, the elbow and the shoulder-joint. The contrast in the condition of the two arms in this respect was striking; the patient recognised that he was entirely ignorant of the position of his left arm in space, whereas every movement of any part of the right arm was immediately recognised.

Such were the local effects produced by a tumour growing in the left half of the spinal cord. The remote effects were shown by profound sensory changes in the right half of the body, the right leg and right arm below the level of the fourth cervical segment. Within this area, he was insensitive to pain, to heat and to cold, but light touches and pressure were everywhere appreciated, and localised with accuracy. The sense of passive movement and position was disturbed in the left foot, that is, on the same side as the maximum lesion, but on the opposite side to the analgesia and thermo-anæsthesia.

This case shows, that a growth which destroyed the whole of the left half of the spinal cord produced complete analgesia and thermo-anæsthesia in the left arm at the level of the lesion. The remote loss of sensation to pain and temperature lay over the right or opposite half of the body. Moreover, the sense of passive position and movement was disturbed in the left or analgesic arm, in consequence of the local effect of the growth; whereas its remote effect was manifested by a loss of the sense of passive position and movement in the left foot. This was accompanied by total analgesia and thermo-anæsthesia of the right half of the body, below the level of the fourth cervical segment Sensibility to touch and pressure remained unaltered until late in the disease. Finally, even this form of sensation was disturbed in the left arm.

Syringomyelia rarely disturbs all forms of sensation at the level of the lesion, but, as far as it occurs, the loss of sensation follows the rules that can be deduced from Case 10. So long as the symptoms remain unilateral, the loss of sensation will be found on the same side as the muscular

wasting. This is so well recognised, that we need only mention shortly some cases among our collection which illustrated this rule.

In April, 1905, Walter J. H. (Case 12) was an excellent instance of syringomyelia with unilateral manifestations. Sensibility to pain and temperature was lost over the whole of the right forequarter, but no other form of sensation was disturbed. Neither hand was wasted and there was no loss of motor power. By August, 1906, wasting and loss of power had appeared in the right hand.

Similarly, Florence R. (Case 14), when first examined in 1899, showed distinct wasting and loss of power of the left hand, accompanied by thermo-anæsthesia, confined to the preaxial half of the left forearm and hand. Shortly afterwards, wasting of the right hand was noticed; but even as late as March, 1902, the loss of sensation was still confined to the left forearm and hand; but in November, 1902, part of the right hand also became insensitive to heat and cold. Here, again, the loss of sensation appeared on the same side as the loss of motor power and muscular wasting.

Such cases are examples of the rule that the local manifestations of intramedullary disease are found upon the same side as the disturbance of motion and the wasting of muscles.

Section 2.—The Nature of the Local Disturbances of Sensation produced by an Intramedullary Lesion.

In Chapter 3, we contrasted the nature of the loss of sensation due to destruction of peripheral nerves with that caused by a lesion of the spinal cord, choosing as an instance the phenomena of Brown-Séquard paralysis. From such a comparison, it became possible to formulate certain fundamental differences between the sensory loss in the two cases. We shall now attempt the more difficult task, and compare the characteristics of the loss of sensation, due to the local activity of an intramedullary lesion, with its remote sensory effects. The manner of response to each particular stimulus will be detailed in order, and we shall indicate the similarity and differences in the loss of sensation produced by the two conditions.

(*a*) *Heat and Cold.*

As is well known from the phenomena of syringomyelia, sensibility to pain, heat and cold may be lost without any other sensory defect. Any one of these forms of sensation may be disturbed separately, or to a varying amount, and it is a commonplace of neurology that thermo-anæsthesia may be the only sensory concomitant of this disease. Moreover, sensibility to heat is not infrequently more widely destroyed than that to cold. It is certain, therefore, that the local manifestations of an intramedullary lesion may disturb sensibility to heat, and leave sensibility to cold unaffected. C. H. (Case 11) was an instance of this condition; at one period of his illness, a large portion of his face was insensitive to heat though sensitive to stimulation with cold.

The converse condition may also be present, and the extent of the loss of sensibility to cold may exceed the loss to stimulation with heat.

But, ultimately, if the disease extends, the borders of the loss of sensation to heat will generally become coterminous with those of the loss to cold. The isolated anæsthesia to one form of temperature stimulation is of interest, solely as showing that a local lesion can disturb the one form of sensibility over parts that remain sensitive to temperatures at the opposite end of the scale. In this, the local manifestations of an intramedullary lesion agree with its remote effects described in Chapter III., and differ fundamentally from the loss of sensation caused by destruction of peripheral nerves.

With regard to the character of this loss of sensibility to heat, it is important to discover whether sensation is simultaneously disturbed for all temperatures we call warm or hot. This is frequently difficult to determine. For the borders of an area insensitive to thermal stimulation frequently merge gradually into parts of normal sensibility; here a temperature of 50° C. will cause a sensation of heat, although the weaker stimulus of 40° C. cannot produce an impulse of sufficient intensity to reach the sensorium. In the same way, temperatures of 22° C. may produce no sensation, although coldness is easily evoked by stimula-

tion with ice. Here the sensibility of the part to heat and to cold is lowered, and the intermediate degrees of temperature therefore become stimuli of insufficient intensity.

We have never been able to assure ourselves in any instance that, in consequence of a pure intramedullary lesion, the patient was sensitive to intermediate degrees of heat and cold but insensitive to extremes.

When the thermo-anæsthesia has remained stationary for a long period, so that this border of partial loss of sensation has disappeared, it is not difficult to show that sensibility to heat is destroyed as a whole. In a similar manner, it can be shown that over areas insensitive to cold, no temperature between ice and 24° C. produces any sensation of coldness. In Case 14, careful experiments, made on September 10, 1906, and on many previous occasions, showed that sensibility to all degrees of heat and cold was destroyed over the left half of the face bounded by the middle line. Ice, water at 22° C., at 38° C., at 40° C., and at 50° C. were all called "a touch" over the left half of the face; whereas, when the middle line was passed, they were accurately discriminated and rightly named.

A similar condition was seen in W. J. H. (Case 12, Fig. 31) at the frontal border of the loss of sensation. On a photograph taken in April, 1905, this border corresponded exactly in shape and position with that marked out in September, 1906, showing that the loss of sensation had ceased to extend in this direction. At this border, no temperature at any degree between ice and 60° C. produced any other sensation but that of touch. In this case, the abruptness of the boundary between parts of normal and abnormal sensibility made it unusually easy to determine this fact.

But it is not only in syringomyelia that the local manifestations of an intramedullary lesion may produce a thermo-anæsthesia, which conforms to this description. A rapidly advancing growth may cause a similar loss of sensation. Parts become insensitive to heat which are sensitive to cold, and no response may be evoked by stimulation with any degree of temperature which normally produces a sensation of heat. This was well shown by the case already

cited on p. 587 (No. 9), where a tumour growing mainly in the left half of the spinal cord disturbed sensation in the left arm and right half of the body. Here we had the opportunity of comparing the local thermo-anæsthesia on the side of the lesion with that on the opposite half of the body due to its remote manifestations; and in both cases, the character of the loss of sensation was the same. The left arm, equally with the right leg, was insensitive to all temperature stimuli.

To sum up, therefore: (1) the local action of a lesion within the spinal cord may produce a more extreme thermo-anæsthesia to heat than to cold, or *vice versâ*. Parts sensitive to the one end of the scale may be insensitive to temperatures at the other end.

(2) We have found that an area insensitive to heat will also be insensitive to all temperatures that produce a warm sensation on the normal skin. Towards the edges of the thermo-anæsthetic areas, water at 40° C. may produce no sensation, although 50° C. is recognised as hot. This is due solely to the greater intensity of the stimulus of the higher temperature over parts of defective sensibility; it does not occur when the loss of sensation has remained stationary for long periods.

(*b*) *Pain.*

In every instance of Brown-Séquard paralysis where the patient was insensitive to cutaneous painful stimuli, he was also insensitive to the pain of deep pressure, although its gradual increase could be perfectly recognised. This characteristic association between cutaneous analgesia and loss of sensibility to the pain of deep pressure, is also found as one of the local manifestations of an intramedullary lesion.

Thomas B. (Case 13) is an excellent instance of this association. He was insensitive to the pain of a prick and of the interrupted current over both upper extremities and the upper half of the trunk (Fig. 6, p. 556). Over the whole of this area, pressure failed to cause pain, even when raised to 15 kgm.; and yet he was nowhere insensitive to deep touch, and recognised the gradually increasing pressure.

Elsewhere, outside the analgesic area, a pressure of from 3 to 5 kgm. invariably caused pain.

A similar condition was found in Case 12. Here the whole of the right upper extremity and the right half of the cervical and thoracic regions of the trunk were insensitive to the pain of a prick and the painful interrupted current (Figs. 21 and 22). Sensibility to touch, both superficial and deep, was everywhere perfect; but, over the whole of the right upper extremity, an increase of pressure up to 15 kgm. caused no pain. When we first saw him, the right upper extremity only was analgesic (Fig. 21). But

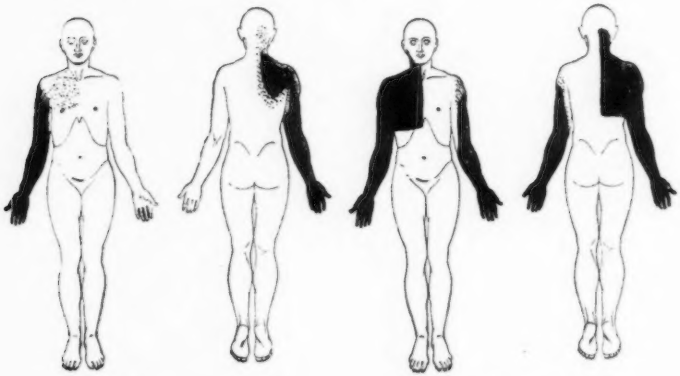


FIG. 21. FIG. 22.

To show the loss of cutaneous sensibility to painful stimuli in Case 12. Fig. 21 shows the extent of the analgesia in 1905, fig. 22 the parts affected in 1906.

later, the left hand and forearm also became insensitive to prick, and the loss of sensation is evidently advancing in this limb (Fig. 22). Here his behaviour when tested with the algometer was instructive. The loss of sensation to prick and to the interrupted current extended to the left elbow and at times even above that point. Over the palm, a pressure of 4·5 kgm. was painful, and he complained when the pressure over the flexor aspect of the forearm reached 5 kgm. Although the forearm and hand were insensitive to prick and to the pain of an interrupted

current, pain could still be produced by pressure little in excess of the normal.

Thus, over parts where the cutaneous analgesia has been recently acquired, increasing pressure not uncommonly produces a painful sensation. But, over an old-standing area of cutaneous analgesia produced by the local effects of an intramedullary lesion, pressure no longer produces pain; and yet sensibility to the tactile element of pressure remains entirely unaffected.

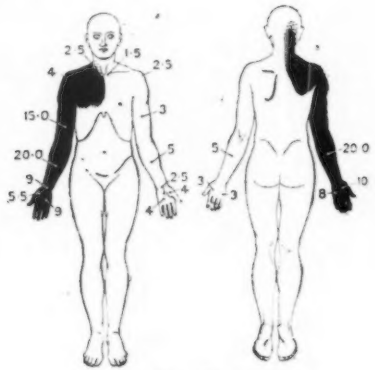


FIG. 23.

To show the loss of painful sensibility in Case 11 (Appendix, p. 698). The area marked in black corresponds to the extent of the cutaneous analgesia. At various points are placed numbers corresponding to the pressure which caused pain. Thus 5·5 means that pain was caused by an algometer pressure of 5·5 kilograms. But such a number as 20=0 means that no pain could be caused even by a pressure of 20 kilograms.

This persistence of sensibility to the pain of pressure over parts that correspond to the outworks of an advancing lesion, can lead to the following anomaly. The skin of the whole upper extremity may be insensitive to all painful stimuli, although pain can be produced by pressure over the palm of the hand. In Case 11, this phenomenon was the more striking in that the right arm and hand were entirely unaffected, and could be used for comparison with the analgesic extremity. On Fig 23, is shown the extent of the cutaneous analgesia, and at each point is given the

pressure in kgm. which produced pain. It is at once evident that the whole forearm, back and front, was insensitive to both cutaneous and deep pain. But, over the right palm, a pressure of 5·5 kgm. still produced pain; over a similar part of the normal palm, pain was evoked by a pressure of 4 kgm., a difference within the range of experimental error. Here the whole of the forearm on the extensor and flexor surfaces was insensitive both to painful cutaneous stimuli and to the pain of deep pressure; but, to the latter, the palm remained sensitive,

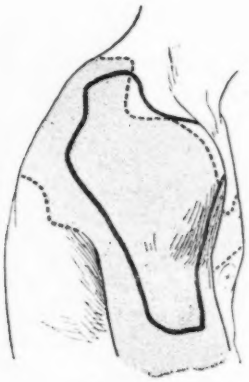


FIG. 21.

To show the relation of the deep hyperalgesia to the cutaneous analgesia in Case 11 (Appendix, p. 698). The area of cutaneous analgesia is coloured gray and enclosed by a broken line; the area hyperalgesic even to the lightest pressure is enclosed by a heavy black line.

though insensitive to cutaneous painful stimulation. This is evidently due to a difference in the segmental distribution of the afferent mechanism employed in the conduction of painful impulses produced by cutaneous and by deep stimulation. The pain produced by deep pressure on the palm is conducted by fibres which run with the muscular nerves. It probably enters posterior roots other than those which receive the afferent impulses from the skin of the same parts. If this were so, the impulses from the termination

of the limb would probably enter those segments which correspond to the motor supply of the part. Now, the hand receives its motor innervation from the first thoracic and eighth cervical. In the case we have quoted, these segments, judging by the cutaneous loss of sensibility, would lie at the edge of the active lesion in the cord, and the hand would remain sensitive to the pain of deep touch, because it was represented by parts not in the focus of destruction.

This want of correspondence between the extent of the cutaneous and deep analgesia, produced by the local manifestations of an intramedullary lesion, led to the following extraordinary condition, in the case we have just considered (No. 11, Fig. 24). The skin of the whole upper extremity was analgesic to the prick of a pin and to the painful interrupted current. This man, however, suffered from a neuropathic destruction of the right shoulder-joint (Charcot's joint). This joint was excessively painful, and, over a considerable area enclosed within the thick black line on Fig. 22, he was profoundly hyperalgesic to even the lightest pressure; even pressure with the head of a pin caused him to cry out. At the time when this area of deep hyperalgesia could be marked out, the whole of the parts tinted gray on Fig. 24, were insensitive to all cutaneous painful stimuli. That is to say, there was a large area of the arm and shoulder abnormally tender to pressure, though the skin was entirely insensitive to the prick of a pin. Ultimately this hyperalgesia disappeared with the gradual advance of the loss of sensation. (For further details, vide Appendix, p. 702.)

In conclusion, we find that the analgesia, produced by the local manifestations of an intraspinal lesion, consists essentially of insensibility to the pain of both cutaneous and deep stimulation. This loss of sensation to painful pressure exists without any disturbance of sensibility to its tactile element; the patient not only knows he is pressed, but recognises the gradually increasing pressure.

In these characters, the analgesia produced by the local manifestations of intramedullary disease corresponds exactly with that due to its remote effects.

(c) *Touch and Pressure.*

One of the most striking differences between the form assumed by the loss of sensation in Brown-Séquard paralysis and that which follows injury to peripheral nerves, lies in the behaviour of the patient to the stimuli of superficial and deep touch. If sensibility to touch is disturbed in Brown-Séquard paralysis, the patient no longer responds to either form of stimulation.

Loss of tactile sensibility is a rare local manifestation of syringomyelia; but we are able to bring forward two

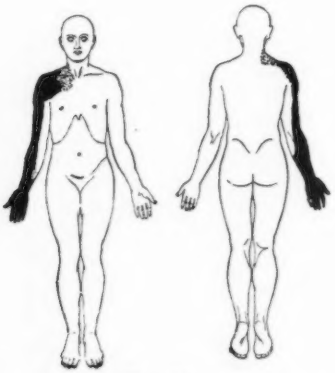


FIG. 25.

To show the loss of tactile sensibility in George B. (Case 15, Appendix, p. 720). Over the area marked black he was insensitive to all forms of tactile stimuli, superficial and deep; over the dotted area he was insensitive to light touch, but sensibility to deep touch, though present, was certainly somewhat diminished.

instances from our collection. From these cases, it would seem that the form assumed by the loss of tactile sensibility corresponds exactly with that of the remote tactile anæsthesia in Brown-Séquard paralysis. Sensibility to light touch and sensibility to pressure were lost over almost exactly the same extent.

Fig. 25 shows the area insensitive to light touch and to pressure in the case of George B. (No. 15). For seven

years he had lost sensation in the right hand, accompanied by a gradually increasing inability to write. In October, 1906, the sensations of pain, heat and cold were lost over the right upper extremity and the greater part of the right half of the head and neck. Sensibility to pressure was destroyed over the area marked in black on Fig. 25; over this area, together with the dotted parts, he was entirely insensitive to light touch. These two areas corresponded closely, except at the extreme frontal border, where the extent of the loss of sensation to light touch somewhat exceeded that of the loss to pressure. But, since stimulation with No. 8 of von Frey's hairs no longer produced any sensation when the border of loss to light touch was passed, we may assume that deep sensibility was also diminished. Pressure caused a sensation only by its greater intensity as a tactile stimulus. On the inner aspect of the arm, the borders of the anæsthesia to the two forms of tactile stimulation coincided almost exactly.

The second case where tactile sensibility was disturbed as a local manifestation of intramedullary disease, is that of R. A. H. (No. 16, Appendix, p. 727). When this man was first seen in March, 1904, all loss of sensation was confined to the left arm and neck, except that to cold, which was bilateral. Tactile sensibility was completely abolished over the whole of the left upper extremity. The motor disturbance was represented by paralysis of the left vocal cord and the left half of the palate. The left knee jerk was greatly increased, clonus was obtained from the ankle and, on the same side, the plantar reflex gave an extensor response.

Thus it would seem that, in those rare instances where syringomyelia produces loss of sensation to touch, it also causes corresponding anæsthesia to the tactile element of pressure.

More rapid growths, starting within the spinal cord, frequently spread to the posterior roots, or produce secondary metastases which become extramedullary. This makes the results obtained from such malignant growths extremely unsatisfactory. Many of the sensory phenomena

they cause are of mixed origin, and not due solely to intramedullary disease.

As far as our material goes, we believe that, when tactile sensibility is disturbed at the level of the lesion in consequence of intramedullary disease, the part becomes insensitive to stimulation with touch, both superficial and deep. Towards the extreme edges, pressure may be appreciated, but the skin may be insensitive to the lighter stimulus; this is due solely to difference in the intensity of the stimulus, and accurate tests will show that, even over this borderland, sensibility to deep touch is diminished.

Thus, the condition of tactile sensibility at the level of the lesion closely resembles that due to its remote effects.

(*d*) *Passive Movement and the Discrimination of Two Points.*

Among the distant manifestations of an intramedullary lesion, any loss in the sense of passive position and movement was always found to be associated with defective answers to the compass-test. (Chapter III., Section 3, 4.) A similar relation is found, when this form of sensation is disturbed at the level of the lesion. So long as the disease remains purely intramedullary, there is not that close association between the discrimination of two points and sensibility to light touch usually found after lesions of peripheral nerves or posterior roots; nor is the power of recognising passive movement and position associated with the integrity of deep touch.

Of this we can bring forward one unusually perfect example. Joseph F. (Case 9) suffered from a glio-sarcoma, which destroyed the spinal cord in the cervical region more extensively on the left side than the right. He was unable to recognise the position of any part of the left upper extremity, and no passive movement of the joints, however extensive, produced any sensation. And yet, at first, sensibility to both superficial and deep touch was unaffected.

Over the left hand, he was unable to discriminate two points of the compasses when separated for a distance of 5 cm.; over the right hand he made two false answers only at 1 cm., and at 2 cm. he was invariably correct.

Right hand 1 cm.	1	9 R. 1 W.	Left hand 5 cm.	1	10 R.
	2	9 R. 1 W.		2	10 W.
2 cm.	1	10 R.			
	2	10 R.			

Here the power of discriminating two points was profoundly impaired, although tactile sensibility was at that time unaffected, and the sense of passive movement and position was lost, in spite of the retention of sensibility to pressure.

Thus, it would seem that, when the loss of sensation at the level of an intramedullary lesion includes disturbance of the sense of passive position and movement, the discrimination of two points is also affected.

(*e*) *Summary.*

A survey of the character assumed by the loss of sensation at the level of a pure intramedullary lesion of the spinal cord has led to the conclusion, that this loss follows the central rather than the peripheral type, although it is situated in those parts which lie on the same side as the disease. If sensibility to heat is disturbed, the patient no longer recognises temperatures between 30° C. and 60° C. But, over the same parts, he may be sensitive to all temperatures which normally produce a sensation of cold. Where the skin is insensitive to painful stimuli, deep pressure will no longer be painful. Sensations of light touch and the tactile aspect of pressure are linked together, so that the former is a less, and the latter a more intense, degree of tactile stimulation. When the patient becomes unable to recognise the position of his limb, assumed passively, it will be found that he is also unable to discriminate two points at distances much in excess of that normally requisite.

CHAPTER V.

Afferent Segmentation within the Spinal Cord.

Whenever several observers, working on the same problem, bring out results which differ greatly, in spite of fundamental similarity, it is certain they have adopted

different methods. The present state of text-book knowledge on segmentation of the central nervous system is an instance of the confusion which may arise from an attempt to combine the results of different observers, without recognising the significance of the methods by which they have been obtained. (Seiffer [27], Lewandowsky [17], Oppenheim [20]).

Those who have worked on the segmentation of the afferent nervous system from their own observations, and by original methods, can be classified into the following groups.

(*A*) *Distribution of the Posterior Roots.*

(1) Sherrington [30] determined the total afferent distribution of each posterior root, by the method of residual sensibility. By dividing a number of posterior roots above and below the one nerve root under examination, he left a sensitive area surrounded on all sides by anæsthesia. This sensitive area must have been innervated by the one remaining posterior root. But, of the specific function of these afferent nerve fibres, he remained of necessity ignorant. His areas correspond to the maximal extent of the fibres of one posterior root, whether they be those of epicritic, of protopathic or of deep sensibility. We shall therefore speak of the areas worked out by Sherrington as maximal root areas.

(2) Head and Campbell [12] recorded the areas on the surface of the body mapped out by the eruption of herpes zoster. They then determined in seventeen instances by *post-mortem* examination which posterior root ganglion was the seat of the disease. Now, it is obvious that, if the whole of one ganglion is affected, the extent of the eruption will correspond to the distribution of those fibres only which, when irritated, can cause vaso-dilatation and destructive lesions on the skin. Head and Sherren [13] have shown reason to believe that such fibres are closely associated with the protopathic system. (*Cf.* also Bayliss [1]).

It is therefore probable, that the areas marked out by herpes zoster correspond to the segmental arrangement of

the protopathic fibres as they pass through the posterior root ganglion.

(*B*) *Segmentation within the Spinal Cord.*

(1) As revealed by irritation (Hyperalgesia).

Head [11] worked out a scheme of intramedullary segmentation based on the hyperalgesic zones which arise in consequence of visceral irritation. Disease of an internal organ produces impulses which pass into the spinal cord by the white rami. The segment upon which they impinge is excited, and pain is produced. But, at the same time, all potentially painful impulses passing into this segment from the somatic afferent nerves are exaggerated, and ultimately the body wall may become tender.

These hyperalgesic zones represent the extreme projection on the surface of the body of those segments within the spinal cord which receive painful impulses from the periphery.

(2) As revealed by loss of sensation due to intramedullary lesions (Kocher [15], Allen Starr [33 and 34], Thorburn [35 and 36], Wichmann [40]).

Of these observers, Kocher alone faced the difficulty and determined to record the upper level of the analgesia in a large number of cases of verified spinal lesions. That part of Wichmann's valuable monograph which deals with the spinal cord is also based on the loss of sensation, produced by intramedullary disease; but, in his general scheme, he attempts to combine the results obtained by other means. Thorburn assumed that the areas of loss of sensation corresponded to the form and extent of the supply of segments within the spinal cord, and failed to appreciate that they represented those parts only, which were not supplied from the lowest intact segment. Moreover, he failed to differentiate between the loss of sensation due to destruction of roots as they enter the spinal cord and that of intramedullary segments.

A want of appreciation of the different methods employed by these observers, and the non-recognition of the fact that

their results applied to different levels of the nervous system, have led to endless confusion.

The appearance of Sherrington's work on the posterior roots caused clinicians to revise their views with regard to the distribution of sensory disturbances in certain well-known diseases such as tabes dorsalis. Max Laehr [16] was the first to recognise that the loss of sensation in syringomyelia was distributed segmentally, at a time when most observers believed that its arrangement more nearly corresponded to that found in hysteria. Since then, many clinicians have acknowledged the truth of his observations and have amplified his statement in various directions.

Clinical observers have relied on the extent of the loss of sensation in their attempts to prove the segmental distribution of a lesion within the spinal cord. All would have been well, had they correlated their results solely with the borders of the loss of sensation in verified cases of interruption of the cord at various levels. But the attempt to compare these areas of loss of sensation from intra-medullary disease with the maximum supply of posterior roots led to nothing but confusion.

No clinician seems to have recognised, that the complete cutaneous distribution of a nerve, of a posterior root or of a segment of the spinal cord, could only be discovered by observing how much of the skin was sensitive, when that afferent path alone remained intact. If we wish to know how much of the palm is supplied by the median nerve, we cannot do so by observing the extent of the loss of sensation caused by its division. A case must be selected where the ulnar, the radial and the external cutaneous nerves have been divided; then, any part of the hand which remains sensitive must receive its innervation from the median. (*Cf.* Head and Sherren [13].)

This is the method of residual sensibility invented by Sherrington, and so ably employed by him to discover the maximum afferent distribution of each posterior root.

If we wish to determine the exact level of the affected intramedullary segment, we are compelled to adopt an indirect method. For lesions within the spinal cord, such as those produced by fracture of the spine or rapidly spreading growths, lead to wide-spread destruction. However well defined may have been the disturbance of sensation, some disease will be found corresponding to parts beyond its limits. Moreover, the opportunities are too limited for correlating these areas of residual sensibility with the extent of intramedullary disease.

Head [11] showed that there is a close correspondence between the distribution of the tenderness caused by irritation of the segments within the spinal cord, concerned with the reception of painful impulses, and the areas marked out on the skin by the eruption of herpes zoster. Now, herpes zoster is due, in most cases, to acute inflammation of a posterior root ganglion, whereas the tender areas in visceral disease are due to the irritation of intramedullary segments. But, though all the cells and the fibres peculiar to them must be affected by the profound inflammation of the ganglion, one system only, as far as we know, can produce an antidromal effect upon the skin. These are the fibres shown by Bayliss [1] to have their cells of origin in the ganglion of the posterior root. Bayliss, in conjunction with one of us, has been able to show, that these antidromal fibres are capable of excitation in the divided nerve of a cat, five weeks after it has been reunited to the central nervous system. In the time and manner of their regeneration, these fibres closely resemble those of the protopathic system. Thus, it is probable that this power of producing acute changes in the skin of the periphery is a function of fibres, which run in the protopathic system.

On this hypothesis, the eruption of herpes zoster would correspond to the distribution of the fibres and cells of the protopathic system in the ganglion of the posterior root.

Now Head and Sherren [13] showed, that division of several posterior nerve roots caused a more extensive loss of protopathic than of epicritic sensibility. Expressed

crudely, sensibility to painful cutaneous stimuli was lost over a larger area than sensibility to light touch. From this fact alone it is certain, that the protopathic fibres in the posterior roots supply areas which overlap one another to a less extent than those of the epicritic system.

It is therefore not surprising to find a comparatively close correspondence between the form and extent of the areas marked out by herpes zoster, and the hyperalgesic zones of visceral disease. They belong, it is true, to different levels of the nervous system; the herpetic zones correspond to the distribution of those fibres which conduct all the cutaneous painful impulses, whilst the others represent the segments of the spinal cord into which those impulses impinge.

Since it is impossible to obtain enough material to determine the enumeration of the segments by means of destructive lesions of the spinal cord, it is justifiable to turn to the known levels in herpes zoster. For, since the seventeen *post-mortem* examinations by Head and Campbell, it has become possible to determine the site of the lesion with precision, in most cases of this disease. Even should it ultimately turn out that the likeness between the areas of tenderness in visceral disease and those of herpes zoster has been exaggerated, we can still determine the numerical level of an intramedullary segment, by comparing the extent of the residual sensibility with the areas marked out by herpes zoster.

(*a*) *The Sensory Disturbance at the Level of the Lesion shows signs of Intramedullary Segmentation.*

By applying this method of residual sensibility to cases where the sensory impulses have been interrupted by disease within the spinal cord, it would be possible to arrive at a notion of the caudal borders of the intramedullary segments for touch, for pain, for heat and for cold. In every case, the border must be determined at which sensation to each of these stimuli ceases.

But, unfortunately, in an overwhelming majority of cases, the boundary between parts of normal and abnormal sensibility is ill defined. The upper border of the anæs-

thesia for any one stimulus may differ in position according to the method of testing. If, for instance, we start from parts of normal sensibility, the patient's attention is awakened, and he will respond to the stimulus even within areas which would appear to be insensitive if the testing began in the reverse direction. The border obtained by passing from normal to abnormal parts may differ greatly from that at which sensation is present, when we travel in the reverse direction. If so, it is certain that the specific

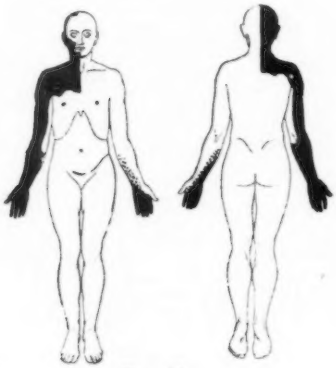


FIG. 26.

To show the loss of sensation to cutaneous painful stimuli in Case 15. (Appendix, p. 720.)

Note the area of residual sensibility on the inner aspect of the right arm; its frontal border corresponds to that of the 2nd thoracic segment.

sensation we are testing is diminished, but not absolutely lost, over an area which extends between the two borders. Such cases are useless for determining the form and position of intramedullary segmental borders.[1]

In order that an area of disturbed sensibility can be used to investigate intramedullary segmentation, the position of the border of residual sensibility must remain constant from day to day. Moreover, the border at which the specific

[1] For an instance of such a border, *cf.* Case 6, p. 674.

sensibility ceases must correspond closely with that at which it begins when testing in the opposite direction.

Unfortunately, such well-defined areas of total loss of sensation are rare manifestations of intramedullary disease. But they occur sufficiently frequently for us to be certain that the boundary between sensitive and insensitive parts corresponds to the border of an intramedullary segment. For instance, in Case 15, an area on the inner aspect of the right arm alone remained sensitive to cutaneous painful stimuli (Fig. 26). The border of this residual sensibility should correspond to an inter-segmental boundary.

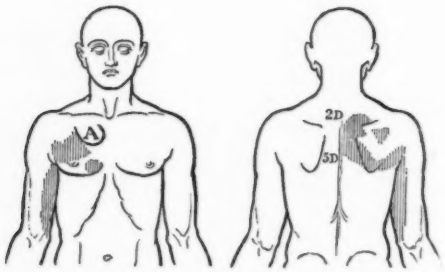


FIG. 27.

To show the area of superficial tenderness due to an aneurysm of the aorta. The frontal border of this area corresponds to that of the 2nd thoracic segment.

Now, the only scheme of intramedullary segmentation based on any method except loss of sensation is that of the hyperalgesic zones in visceral disease. Compare the area of residual sensibility on Fig. 26 with the tenderness found in a case of aortic disease (Fig. 27). It will be seen how closely the extent of the one corresponds with that of the other.

From a case of herpes zoster examined after death (Head and Campbell, Case 7), we know that this area of residual sensibility must have corresponded to the distribution of the second thoracic segment (Fig. 28).

Another boundary, which remained over a considerable

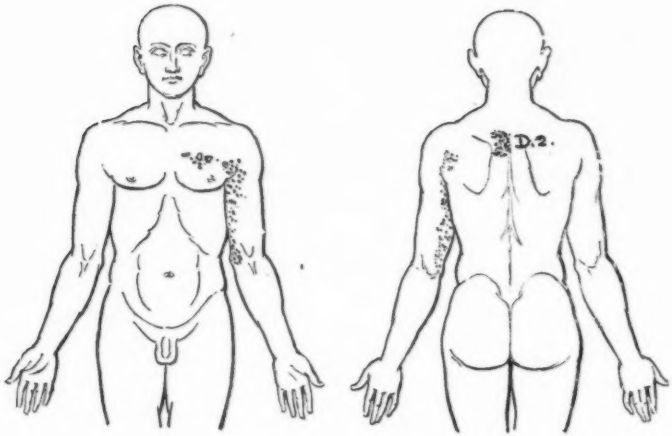


FIG. 28.

To show the distribution of the eruption in a case of herpes zoster, proved by *post-mortem* examination to be due to inflammation of the ganglion of the 2nd thoracic posterior root. (Head and Campbell [12], Case 7.)

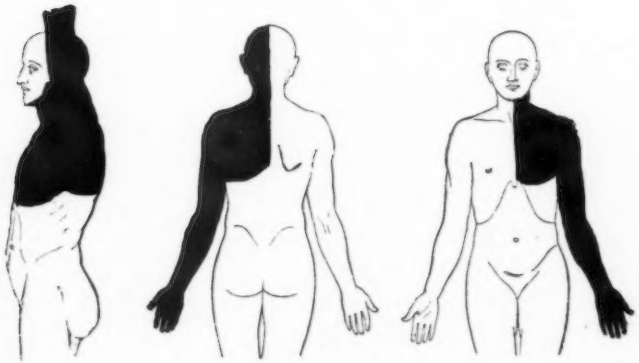


FIG. 29.

To show a well-defined boundary between parts insensitive to prick and those of normal sensibility. (Case 16, Appendix, p. 727.)
The boundary of the residual sensibility corresponds to the frontal border of the 7th thoracic segment.

period constant to cutaneous painful stimuli, is shown on Fig. 29. This was taken from a case of syringomyelia (Case 16), where the disease affected mainly the medulla and cervical spinal cord.

Compare this intramedullary segmental area with the distribution of herpes zoster in a case where the ganglion of the seventh thoracic posterior root was diseased (Fig. 30). It will be seen how closely the frontal border of this eruption corresponds to the boundary of residual sensibility to painful stimulation in Fig. 29.

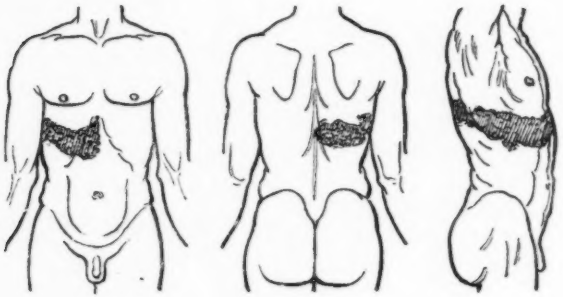


FIG. 30.

To show the distribution of the eruption in a case of herpes zoster proved by *post-mortem* examination to have been due to inflammation of the ganglion of the 7th posterior root. (Head and Campbell, Case 2.)

We have compared the border of an area of residual sensibility with the border of a zone of hyperalgesia, due to the disturbance of an intramedullary segment by visceral irritation. But a glance at Figs. 26, 29 will show that the segmental nature of the disturbance in the two instances of syringomyelia could have been deduced even from an examination of the extent of the analgesia, for the boundaries of the area insensitive to painful stimulation corresponded closely with that of the parts sensitive to the same stimuli. Thus, neighbouring segmental zones cannot overlap one another extensively on the trunk. The amount to which the distribution of one segment encroaches

on that of its neighbours is probably less than one half its own extent. Thus it comes about that even the loss of sensation to prick, to heat and to cold may closely represent the true segmental distribution of the intramedullary centres.

The frontal border of the loss of sensibility to pain, produced by cutting off all these segments below a certain level, will cause a loss of sensation, that is only slightly lower than the caudal border of the same segment shown by residual sensibility.

For this reason, those observers who have fixed their attention on the cutaneous analgesia and thermo-anæsthesia produced by lesions of several segments of the cord have come closer to the true segmental distribution than would have been the case, had their contention been correct that these segments overlapped greatly.

But this is true only for the loss of sensation to cutaneous painful and thermal stimuli. An intramedullary lesion may manifest itself locally by an area of cutaneous analgesia and thermo-anæsthesia whose borders correspond almost exactly to those at which sensation to pain, heat and cold ceases. But much of this area may be sensitive to deep painful stimuli, such as excessive pressure.

Moreover, tactile sensibility is usually disturbed over a considerably smaller extent, and the borders of this loss of sensation do not correspond to those of the cutaneous analgesia.

It is therefore of fundamental importance, in every case, to test each form of sensation separately, and to record the border at which it ceases and the border at which it begins, when the stimulus travels from sensitive to insensitive areas. If these two borders correspond closely and if the same borders are obtained on several different occasions, it is possible to say that this area may be a segmental border. The form and extent of the boundary of the residual sensibility only will determine if the disturbance of sensation is the expression of intramedullary segmentation.

We have brought sufficient evidence to prove the truth of Max Laehr's [16] original statement, that the phenomena of loss of sensation, caused by intramedullary disease, pointed

to a segmental arrangement of the afferent mechanism at the level of the lesion. But it will be well in the light of this knowledge to examine some of those remarkable borders on the face between parts of normal and abnormal sensibility, so commonly found in cases of syringomyelia.

We shall bring forward such cases only, where these borders remained unaltered for considerable periods. For, when the extent of the loss of sensation is spreading, a single examination may yield results which are transitory, due to the want of adjustment of the higher sensory mechanism to shifting structural changes. But, when a sensory border has remained constant over a long period,

FIG. 31.

FIG. 32.

FIG. 33.

Fig. 31.—From Case 12 (Appendix, p. 705). To show the extent of the residual sensibility to painful and thermal stimulation in 1906.

Fig. 32.—From Case 16 (Appendix, p. 727). To show the extent of the residual sensibility to painful cutaneous stimuli.

FIG. 33.—From Case 14 (Appendix, p. 714). To show the extent of the residual sensibility to painful cutaneous stimuli, taken from a series of observations made between November, 1903, and February, 1905.

as occurs not infrequently in cases of syringomyelia, we can assume that it represents the division between those parts of the central nervous system still capable of function and those in which this power has been destroyed.

The disturbances of sensibility to heat and to cold follow the same segmental arrangement as those for pain. In cases of syringomyelia, it is sometimes possible to find one segmental border marked out by the sensory disturbance to heat, another to cold and a third to pain. Such borders are not, however, usually fixed, and do not therefore come within the subject of this section.

In Case 12, the area insensitive to heat and cold, when passing from normal to abnormal parts, was bounded above by the line on Fig. 31. This was no transitory border, for it has persisted from April, 1905, until October, 1906.

A similar border on the face, associated with a completely different loss of sensibility on the scalp, is seen in Fig. 32. Here the loss of sensibility to cold extended almost as far forwards as the frontal border of the hairy scalp.

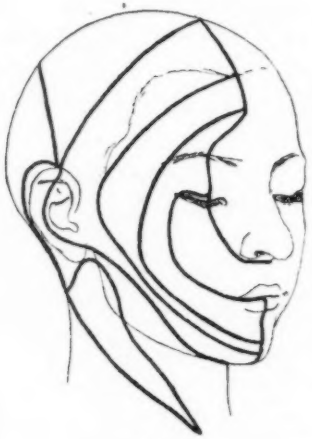


FIG. 34.

Taken from Schlesinger [25] to show the method by which, according to this author, the loss of sensibility advances in lesions of the upper part of the spinal cord and medulla oblongata. Note how closely these borders correspond to those of the tenderness in visceral disease of the organs of the head and face.

Another remarkable area is shown on Fig. 33, taken from Case 14. This was worked out by each of us independently on many separate occasions, between November, 1903, and February, 1905. At this border, all sensibility to cutaneous painful stimuli was lost, and the area over which sensation was present to these stimuli was sharply defined.

We have already shown that on the trunk the residual sensibility to pain, heat and cold tends to follow the dis-

tribution of intramedullary segments. It is therefore probable, that these areas on the face are also distributed segmentally. They certainly do not correspond to the peripheral divisions of the trigeminal nerve.

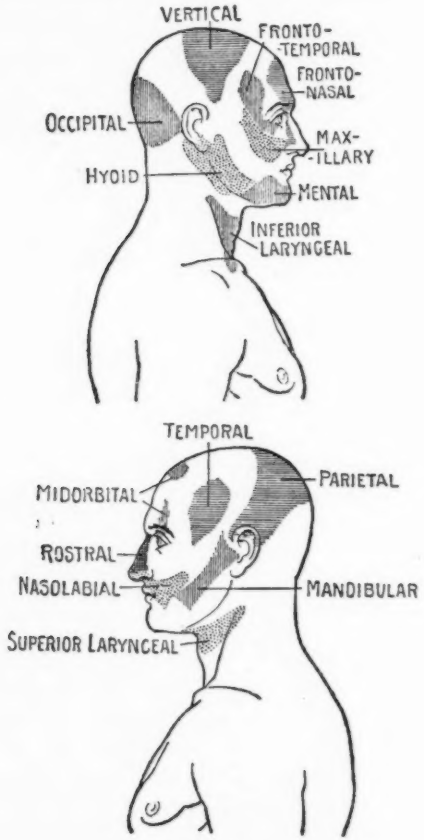


FIG. 35.

To show the extent and distribution of the tender areas which may accompany reflected visceral pain due to disease of the organs of the head and neck. (Taken from Head 11.)

The line on Fig. 31 can scarcely represent the posterior border of residual sensibility of the third division of this

nerve, and it certainly does not correspond to that of its second division. On Figs. 32 and 33, the areas of residual sensibility are inexplicable from the point of view of the peripheral branches of the trigeminal.

The clue to the significance of these areas of residual sensibility is given by the tenderness which accompanies disease of the organs of the head and neck. Fortunately, most of these areas upon the face can be worked out from diseases of the teeth; and, as one tooth only is not infrequently diseased in patients otherwise in perfect health, single areas make their appearance on the face more often than in any other part of the body.

Compare the diagrammatic representation of these areas of tenderness on Fig. 35 with the parts that remained sensitive to pain, to heat or to cold on Figs. 31, 32, 33. It will be obvious, that here lies the key to the otherwise inexplicable distribution of the sensory disturbances of the head and neck, produced by a local intramedullary lesion.

Fig. 31 shows that all segments anterior to the third cervical and hyoid areas were unaffected; but in Fig. 32, the vertical parietal and occipital areas were added to those insensitive in the previous case.

On Fig. 33, it is evident that the only sensitive parts remaining corresponded to the segments in front of the vertical, the temporal and maxillary areas.

Thus, in spite of the difficulties which surround the determination of these tender areas accompanying reflected pain, we find that their distribution alone enables us to understand the forms assumed on the face by the sensory disturbances due to intramedullary disease.

(*b*) *The remote disturbance of sensation on the side opposed to the lesion may show signs of intramedullary segmentation.*

Sensory changes at the level of the lesion might be expected to show signs of a segmental arrangement. But we believe it is also possible to show that, even when the impulses are interrupted after they have passed to the opposite side of the spinal cord, traces of a segmental arrangement can still be discovered.

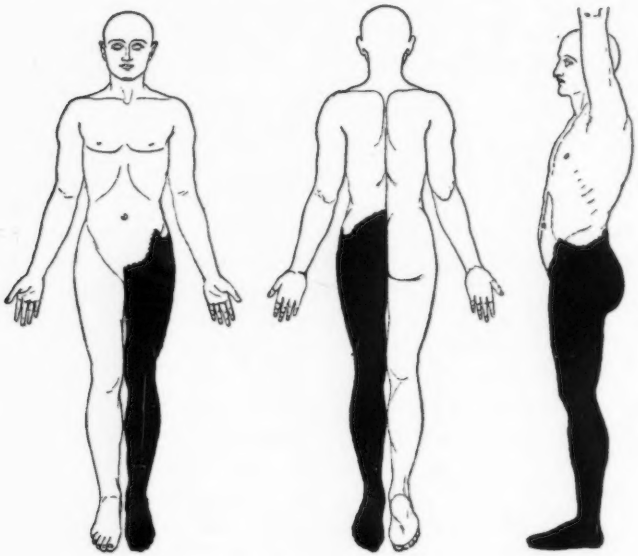


FIG. 36.

To show the disturbance of sensibility to painful and thermal stimulation produced by a lesion which interfered with motion and increased the reflexes in the right leg. (Case 3, Appendix, p. 656.)

The border between the normal parts and those which were completely insensitive was unusually well defined, corresponding to the caudal border of the eleventh thoracic segment.

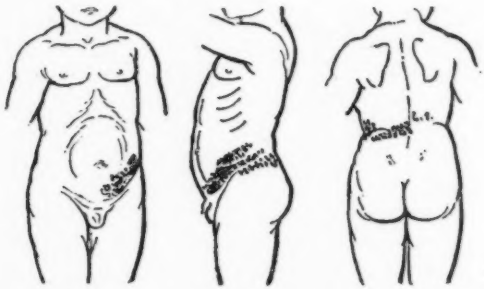


FIG. 37.

To show the distribution of the eruption in a case of Herpes zoster proved by *post-mortem* examination to be due to inflammation of the ganglion of the eleventh thoracic posterior root. (Head and Campbell, Case 5.)

Thus, in Case 3, the border between parts of normal sensibility and those insensitive to pain, heat and cold was well defined on the side opposed to the lesion. This border resembles the caudal limit of the eruption in a case of herpes zoster due to the destruction of the ganglion of the eleventh thoracic posterior root (Fig. 37). Thus, we must assume that in this case the lesion in the left half of the cord interrupted all the painful and thermal impulses which entered on the right side by the twelfth thoracic posterior root, but allowed all those of the eleventh thoracic root to pass unimpeded.

In the same way, in Case 4, an injury of the cervical portion of the spinal cord produced complete loss of sensation to pain, heat and cold over that half of the body opposite to the paralysis of motion. Here also the boundary between parts of normal and disturbed sensibility was well defined, following closely the caudal border of the first thoracic segment. The injury must therefore have destroyed the paths for all those impulses for pain, heat and cold which crossed the cord in the second thoracic segment, and must have destroyed them after the crossing had occurred.

A similar result was produced by a fracture of the third cervical vertebra (Case 2). Motion was disturbed in the right arm, whilst sensibility to heat and cold was entirely abolished over the left leg, left half of the trunk and part of the left arm. The boundary between parts of normal and abnormal sensibility corresponded to the posterior border of an area frequently marked out by herpes zoster, which Head and Campbell attributed to disturbance of the ganglion of the seventh cervical posterior root.

Now, in this case an exploratory operation proved that the neural arch of the third cervical vertebra had been fractured; and yet the anterior border of the remote loss of sensation on the opposite side corresponded to that of the eighth cervical segment. In the same way, the frontal border of the loss of sensation in W. M. (Case 5) corresponded to the parts which we should expect to become analgesic from destruction of the segments below the second thoracic. But there can be little doubt that the upper,

rather than the lower, part of the cervical spine was injured by his fall.

Thus, it would seem that, when the loss of sensation (remote) on the opposite side to the lesion shows evidence of segmentation, it indicates an interruption of the spinal cord, several segments below the point of injury. This was first shown by Piltz [22], whose statements are borne out by our cases of Brown-Séquard paralysis, as far as they go.

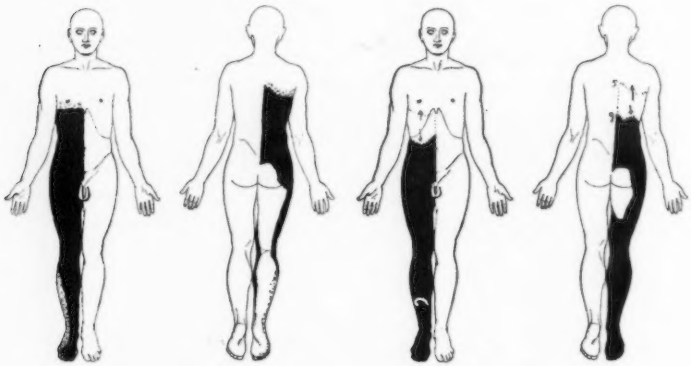


FIG. 38. FIG. 39.

To show the disturbance of sensation produced by injury to the cervical spine in Case 6.

FIG. 38.—Shows the extent of the loss to thermal stimuli. Sensation remained intact over the sacral areas.

FIG. 39.—Shows the extent of the loss to cutaneous painful stimuli. Sensation was present over an area corresponding to the lower sacral segments only.

(*c*) *The remote analgesia and thermo-anæsthesia produced by lesions of the cervical spinal cord, point to a lamellar arrangement of sensory impulses in this region.*

Anyone who has studied a long series of cases of spinal-cord disease will have been struck with the occasional retention of sensation over the sacral areas, in spite of the complete insensibility of the remainder of the trunk. It would seem that the impulses from the caudal end of the

body may escape an interruption which has overtaken all other sensory impulses.

Sometimes this phenomenon also appears in cases of Brown-Séquard paralysis, and it is particularly well seen in the following instances from our collection. The first of these was a man of 41 years who had injured his neck by diving into shallow water, twenty years before he first came under our care. The motor disturbance was represented by distinct wasting of the muscles of the left hand. All loss of sensation was confined to the right leg and right half of the body (Figs. 38 and 39). But, in spite of the widespread cutaneous analgesia, it will be seen that the genitalia with part of the buttock and back of the thigh remained sensitive to prick. The retention of sensibility to heat and cold was even greater, extending to the soles of the feet.

Cyril W. F. (Case 7) showed a similar condition, also produced by diving into shallow water. But, in this case, the loss of sensation was most extensive to heat and of smallest extent to prick (Figs. 9 and 10, p. 559).

It is therefore probable that in the cervical region the impulses from the caudal end of the body (sacral segments), after crossing the cord, pass up in paths separate from those for the sensory impulses from the lumbar and thoracic regions. These paths must be arranged in a lamellar manner, since an injury may interrupt the one set of impulses, leaving the others intact.

Chapter VI.

The Passage through the Spinal Cord of Afferent Impulses concerned with "Localisation."

No question has led to greater difference of opinion than the means by which we recognise the position of our limbs in space, and localise a spot stimulated. Fortunately, in the present paper, we are concerned with the passage through the spinal cord of certain elementary afferent impulses, and not with the question of spacial perception as a whole. This remains a function of the nervous system at a level higher than that with which we are now occupied.

But, throughout this paper, we have spoken of the power of recognising passive position and movement. Recognition of voluntary movement is a more complex process, based upon impulses grouped differently from those which fall within the range of that level of the nervous system we are now considering. We have also examined the power of naming and indicating the position of a spot touched, and, in every case, we have investigated the capacity for discriminating two points at varying distances. These three tests, the recognition of passive position and movement, the discrimination of compass-points and the localisation of a spot touched, have been chosen, because experience shows that they are affected in lesions of the spinal cord.

We have not only avoided all terms relating to the perceptual aspects of localisation, but also those that express the ultimate sum of many streams of diverse sensory impulses, co-ordinated at a higher level of the nervous system than that with which we are now concerned. We shall use the term "tactile localisation" for the power of discovering the spot to which a stimulus is applied. For those impulses which enable us to recognise the two points of the compasses, we shall use the term "tactile discrimination." Of all those impulses which are concerned in the recognition of articular movement, we shall deal with those of passive movement and position only.[1] These are the three spinal afferent elements which at a higher level of the nervous system, together with many other factors, lie at the foundation of spacial localisation.

Section 1. *The Impulses concerned with the Recognition of Passive Position and Movement.*

After division of peripheral nerves, it is found that the power of recognising passive movement and position is closely associated with the integrity of those afferent fibres which run mainly with muscular nerves. It is these fibres

[1] Two of these three terms correspond to the groups classified by German authorities as "Raumsinn" (tactile discrimination); "Hautlocalisation" or "Ortsinn" (tactile localisation): *Cf.* Spearman [32].

which innervate the joints, the tendons and the muscles. So long as they remain intact, all cutaneous nerves may be destroyed, and the patient will still be able to recognise the position into which his joints have been placed. As soon as these fibres are destroyed, this power is lost. But, in the same path as the impulses for the sense of articular movement and position, travel those which underlie the recognition of deep touch, as far as all subcutaneous structures are concerned. The close association between sensibility to deep touch and the recognition of passive movement and position, is best seen by dividing all the cutaneous nerves to any part, without injury to the muscular trunks. The skin is thus rendered totally anæsthetic, and any sensibility which may remain must be due to impulses which travel in the afferent fibres of the deep system running with muscular nerves. It will then be found that the patient can recognise the tactile and painful elements of pressure, and remains sensitive to passive movement and position of every part of the affected limb, although he is insensitive to every other form of stimulation. Injury to peripheral nerves cannot disturb one of these impulses, without simultaneously affecting others of the group. (Head and Sherren [13], Chap. VIII.)

But, in consequence of lesions within the spinal cord, these impulses can be separately affected. The patient may be sensitive to a deep touch, but at the same time all sensations of articular movement and position may be destroyed.

This was the case in J. Y. (No. 10). A secondary growth within the spinal cord had entirely withdrawn from him all knowledge of the position of his legs. So great was the loss of sensation that he was ignorant even of the grossest changes in their position; and yet he was able to recognise pressure everywhere over both legs.

Conversely, in Case 1, all tactile sensibility and all power of recognising differences in pressure were destroyed over the right leg; and yet, there was no part of the limb where he could not recognise passive position and movement. Passive movements were equally well appreciated, whether the joints moved belonged to one limb or the other; and

yet, the right leg was totally insensitive to every other form of stimulation.

With cases of intramedullary disease, it is particularly easy to show that the power of recognising passive articular movement depends upon impulses which are uncrossed and therefore even more elemental than those for pain, heat and cold. For the power of appreciating articular movement and position is always lost on the same side as the disturbance of motion. In Grace G. (Case 3), the left leg was insensitive to all painful and thermal stimulation, but the sense of passive position and movement was perfect. On the right side, sensibility to touch, pain, heat and cold was perfect, but the sense of passive position and movement was disturbed in the right foot. A similar condition existed in Case 2. In consequence of an injury to the cervical spine, this man became insensitive to all painful and thermal stimulation over the left leg and left half of his body. When first seen, the sense of articular movement and position was perfect. But, after an attempt to rectify the condition of his spine, he could no longer recognise the position of his right leg; the surgical operation had destroyed the sense of articular movement and position on the opposite side of his body to the analgesia and thermo-anæsthesia.

This rule, that the sense of articular movement and position is lost on the side of the disturbance of motion, leads to a curious difference between the remote and local sensory phenomena of intramedullary disease. The remote effects consist of loss of sensibility to pain, heat and cold and perhaps to tactile stimuli over the side opposed to the loss of motion; any loss in the sense of articular movement and position will be found in the limbs on the side of the motor disturbance, where the sensibility is otherwise unaffected.

But the local effect of intramedullary disease disturbs cutaneous sensibility on the same side as the motor disturbance, in consequence of an interference with the afferent impulses before they have crossed the cord. The sense of articular movement and position will also be abolished in the same limb, since it is always disturbed on the side of

the loss of motion. This difference is excellently exhibited by the case of Joseph F. (No. 9). This man was analgesic and thermo-anæsthetic over the right leg, in consequence of disease of the cervical spinal cord. The sense of articular movement and position was disturbed in the toes of the left foot, that is, on the opposite leg to the remote loss of sensation to pain, heat and cold. But this growth in the left half of the spinal cord produced a local loss of sensibility to all forms of touch, pain and temperature in the left arm, and, in addition, he was entirely unable to recognise any movement of this arm, however gross.

In conclusion :

(1) The sense of passive position and movement may be destroyed as a consequence of intramedullary disease, although sensibility to deep touch remains perfect. Conversely, the patient may be insensitive to all tactile stimulation, both superficial and deep, and yet may be normally sensitive to passive movements.

(2) Any loss of the sense of passive position and movement will be found on the same side of the body as the disturbance of motion and reflexes.

Thus, the remote effects of intramedullary disease may be manifested in loss of the sense of passive position and movement, disturbance of motion and changes in the reflexes on the one side of the body associated with analgesia, and thermo-anæsthesia of the opposite half.

The local effects of an intramedullary lesion will all be found on the same side; paralysis and wasting of muscles, loss of the sense of passive position and movement, analgesia and thermo-anæsthesia, may all exist together in the same limb.

Section 2.—The Impulses concerned with Tactile Discrimination.

E. H. Weber [39] first employed simultaneous contact with the two points of the compasses as a sensory test. According to him, the power of recognising that a stimulus had an existence external to the body was the highest quality of sensation; sensations grouped together under

the name of "common sensibility" (Gemeingefühl) did not carry with them this quality. He showed that, when the discrimination of two points was used as a test, some parts were many times more sensitive than others; for instance, the two points could not be distinguished over the back, until they were separated to a distance sixty times greater than was necessary for a sensitive surface, such as the tip of the tongue. He believed that, in the compasses, he had discovered a means of measuring the sensibility of the skin, but fully recognised that the test he employed depended upon something apart from crude tactile sensibility.

For, throughout, he speaks of the fineness of the "Ortsinn," and the chapter in which he introduces his experiments is headed "Ortsinn der Haut." His work was imperfectly understood by those who subsequently used the compass-test. Brown-Séquard frankly accepts it as a method of measuring the fineness of tactile sensibility. He does not discuss the method; but, when he wishes to demonstrate the obtuseness to touch of the non-paralysed leg in a case of crossed paralysis, he states that the two points appeared to be one, even when separated to a distance of 13 cm.; on the paralysed leg, the patient discriminated the two points, when they were 1·2 or 1·3 cm. apart. Evidently, in this patient, tactile sensibility was gravely diminished, for Brown-Séquard states that "it was necessary to press very hard" on the left leg, "while the patient was able to distinguish the two points, even when they touched but very slightly the paralysed leg." (Brown-Séquard [7]).

Now it will be obvious, that tactile sensibility may be so gravely diminished, that contact may cause little or no sensation. Under such circumstances, the patient would be unable to recognise the two points, because the contact of one or both failed to produce a sensory impulse. The compass-test would then fail from lack of tactile sensibility.[1]

But early in our research on the sensory results of intramedullary lesions, we found that the power of discriminating two points of the compasses might be disturbed over parts,

[1] *Cf.* Case 4, p. 662.

which were sensitive to every other form of cutaneous stimulation. One leg might be insensitive to painful and thermal stimuli, but remain sensitive in a normal degree to the compass-test; the opposite leg might show a gravely defective sensibility to the compasses, although sensitive to every other form of cutaneous stimulation. We also found that, if tactile sensibility was disturbed, the anæsthesia was found over the leg which was also insensitive to pain and temperature. Three facts stood out clearly before us:—

(1) The power of discriminating two points can be diminished over a limb completely sensitive to all cutaneous stimuli.

(2) If, as a consequence of the remote effects of an intramedullary lesion, the compass-test shows a diminution of sensibility, whilst touch is perfectly appreciated, the part so affected will lie on the same side as the disturbance of motion.

(3) Whenever tactile sensibility is abolished, in consequence of the remote effects of an intramedullary lesion, this loss of sensation will be found on the side opposed to the loss of motion, over the limb insensitive also to painful and thermal stimuli.

It was therefore evident that, although the compass-test depends upon the integrity of tactile sensibility for its existence, it can reveal a disturbance in sensation which, as far as intramedullary impulses are concerned, is entirely separate from sensations of touch. Throughout this work, we have used the compasses as a means of revealing defects in the impulses, which underlie the power of tactile discrimination, apart from a loss of tactile sensibility. That is to say, we have restored this test to the use originally prescribed by E. H. Weber. We have not used it as a test for tactile sensibility as was done by Brown-Séquard, and, by this strict differentiation, we have been able to reconcile the discrepancies which have so puzzled recent observers. A clear understanding of this use of the compass-test is necessary for the comprehension of the results produced by disease within the spinal cord. For if, among the instances where this test showed some loss of sensation, are included those,

where tactile sensibility was otherwise defective, the loss of sensation revealed by the compasses will appear, sometimes on the side of the disturbance of motion, sometimes on the opposite side. But, if we confine ourselves to those cases only, where tactile sensibility was perfect, any defect in discriminating two points will be found to lie invariably on the same side as the disturbance of motion and reflexes. (*Cf.* Case 3, p. 656. Case 16, p. 727).

Section 3.—Tactile Localisation.

We have already considered in this chapter, the effects of an intramedullary lesion on the impulses which underlie the recognition of passive position and movement, and of tactile discrimination. Although they pass to the spinal cord along two afferent paths, these paths become associated within the spinal cord. Not only are the two sets of impulses usually disturbed together by intramedullary lesions, but the loss of sensation to both forms of stimulation is uncrossed, on the same side as the disturbance of motion.

We shall now bring forward evidence to show, that the faculty of tactile localisation is due to impulses which, within the spinal cord, run separately from those of tactile discrimination.

This view was held by Volkmann [38], who first investigated the matter. But the question has been much obscured of late by a misapprehension of the significance to be attached to the compass-test. Some have accepted this test as a measure of tactile localisation, although it was pointed out in 1858, that the compasses tested the "sense of space" (*Raumsinn*) only. In 1901 Förster [8] went so far as to assert, that tactile localisation depended entirely upon sensations of movement (*Bewegungsempfindungen*). However much we may differ from this observer in his general conclusions, he was the first to examine a series of pathological cases from this aspect.

Recently, Förster's challenge has been taken up by an experimental psychologist (Spearman [31]). But from a

clinical point of view, Spearman's case was not completely satisfactory, because the symptoms were evidently bilateral, and sensibility to touch was diminished. We have, however, frequently alluded to this paper, because of the admirable thoroughness of the methods employed.

Before describing in detail the results we have obtained, it is necessary to discuss shortly the methods employed in testing a patient's power of recognising the locality of a spot touched.

First, the patient's eyes are closed and he is asked to say where he is touched. This is the simplest of all methods, and is usually spoken of by us as "spot-naming." If the patient can answer correctly every time he is touched on his great toe, on the heel, on the dorsum of the foot, or on the knee-cap, it is certain that his power of tactile localisation cannot be greatly affected.

If he is asked to point to the spot where he has been touched, he usually errs considerably. But when allowed to grope with the finger he comes closer, and may ultimately touch the very spot. Many observers have pointed out, that this method implies two faculties, a knowledge of the spot touched and of the position in space of the part upon which that spot lies. It requires a combination of the sense of passive position and movement with the power of tactile localisation.

Suppose, however, that the sense of passive position and movement is destroyed, and yet the patient can name correctly every spot touched on the limb. It will then be found, that, if he is allowed to grope for the spot, he fails entirely at first to find the limb, but gradually, as testing proceeds, becomes increasingly able to approximate his finger to the spot. That is to say, by means of the touch and movements of his indicating hand, he has partly corrected his defective knowledge of the position of the limb. When this knowledge has been reacquired, move the limb without allowing him to open his eyes. Then he will grope for the spot touched as badly as ever he did, although he still names it correctly.

Spearman devised the following method for overcoming

this reacquisition of knowledge by means of groping touch. A hole is pierced in a piece of cardboard held one centimetre from the surface to be tested. Through this hole the patient is touched, and he attempts to point out the spot with a pencil. This marks the card, and the distance of the pencil-mark from the hole can be measured. Since the skin is never touched, except by the stimulating object thrust through the hole by the observer, the patient has no opportunity of correcting his defective knowledge by means of touch. This method is an excellent one, where the one limb is affected, and that of the opposite side shows none of the sensory defects with which we are concerned in this chapter. But it only emphasises the double nature of the groping test, which can never be simple so long as we have to deal with the sensory defects produced by an intramedullary lesion.

The power of tactile localisation may remain good over parts where the sense of passive position and movement is entirely absent. In J. Y. (Case 10), who had entirely lost all knowledge of the position of his limbs, we performed the following experiments: His legs were extended in the bed, and he was allowed to see the position into which they had been placed. Then his eyes were closed and he was touched over the sole, instep and just below the knee cap; in every instance his answers were correct, even though the touch was made with cotton wool. With his eyes still closed, the leg was moved into an entirely different position, and his answers were equally correct, although he was entirely ignorant that the leg had been moved, and believed it lay extended before him.

Directly he was asked to point out the spot that had been touched, the double nature of the groping-test was apparent. In the attempt to find the spot he had named correctly, he beat the bed idly, entirely unable even to find his limb.

With the profound disturbance of the power of recognising the position of his limbs was associated an inability to discriminate the compass-points; over the outer surface of the left leg, he failed when they were separated to 15 cm.—

$$15 \text{ cm. } \frac{1 \mid 8 \text{ W. } 2 \text{ R.}}{2 \mid 3 \text{ W. } 7 \text{ R.}}$$

and over the front of the left thigh at 20 cm. distance—

20 cm. $\frac{1 \mid 7\text{ W. }2\text{ R.}}{2 \mid 6\text{ W. }4\text{ R.}}$

But, in spite of this failure to discriminate the two points, he could name accurately the spot touched.

Thus, it is evident that, although the impulses underlying tactile discrimination and the sense of passive position are closely associated in the spinal cord, they must be separated from those of tactile localisation as revealed by the power of naming the spot touched.

R. A. H. (Case 16) also showed that the impulses, which underlie the power of naming the spot touched, pass by paths within the spinal cord, separate from those for the impulses of tactile discrimination. This man showed signs of chronic intramedullary disease affecting the bulb and cervical spinal cord. The motor disturbance was represented by paralysis of the left vocal cord and left half of the palate, with a spastic condition of the left leg.

There was profound loss of the sense of passive position and movement in the left foot and ankle, and tactile discrimination (compasses) was gravely affected. This case was particularly favourable for such tests, because the sensation of the right lower extremity was unaffected and, on the left leg, painful, thermal and tactile sensibility was normal even to the finest tests. On the sole of the right foot his answers were perfect, when the compass points were separated by a distance of 4 cm. Over the left sole, he gave six false answers at 8 cm. $\frac{1 \mid 10\text{ R.}}{2 \mid 4\text{ R. }6\text{ W.}}$

In the same way, when he was tested by moving his left ankle passively, he failed to give a correct answer in thirteen instances out of twenty.

Dorsiflexion	3 W. 6 O. 1 R.
Plantar extension	1 W. 3 O. 6 R.

But, with the right ankle, his answers were quick and uniformly accurate.

And yet, in spite of these grave defects in tactile discrimination and in appreciation of passive position and movement, he named a spot touched as accurately on the left leg as on the right. A number of definite spots on both lower extremities, such as the ball of the great toe, the heel,

the external malleolus, were touched with cotton wool; in no instance did he fail to answer quickly and correctly. Whilst his eyes remained closed, the left foot was placed into an entirely different position. Although he could not tell the situation of the foot, he still named all the points touched as correctly as on the sound side.

But, as soon as he was asked to point out the spot, the difference between the two legs was at once obvious. When tested by Spearman's method, he deviated to the following extent.[1]

	R. (normal).	L. (affected).
Sole of the foot	5·7 cm.	7 cm.
Outer aspect of leg	4·3 cm.	6·8 cm.
Front of thigh	2·5 cm.	4·3 cm.

A further series of experiments were made by stimulating the same spot on the dorsum of the foot four times before and four times after movement, the patient's eyes remaining closed throughout. The results were as follows:—

	R. (normal).	L. (affected).
Before movement	9·7 cm.	9 cm.
After movement	9·7 cm.	13 cm.

It will be seen that the deviation on the sound side remained the same, but on the affected foot had increased greatly after movement.

From these experiments it is evident, that, at the spinal level, the impulses underlying spot-naming run independently of those for tactile discrimination and the recognition of passive position and movement. But, as soon as an attempt is made to indicate the point stimulated, it is necessary that the patient should recognise the position into which his limbs have been placed passively.

At the spinal level, the impulses for tactile localisation seem to be intimately associated with those of tactile sensibility; we have seen no instance where an intramedullary lesion has produced defective localisation of touch without a demonstrable reduction of tactile sensibility.

[1] In this series he was touched six times at each spot; the numbers represent the average deviation.

Section 4.—Summary.

A lesion within the spinal cord can interrupt the afferent impulses which subserve "localisation" in such a way, that the patient fails to answer to one or more of three simple tests. He may not be able to recognise passive position and movement, he may be unable to discriminate two points separated to a distance far in excess of normal, or he may be unable to name the position of a spot stimulated.

We showed reason to believe, that the impulses induced by the first two tests pass up the same side of the spinal cord as that by which they had entered. The remote sensory loss, produced by the interruption of these impulses, even in the cervical region, is found on the side of the motor disturbance.

In this peculiarity, the impulses underlying the recognition of passive position and movement and of the discrimination of two points, differ from every other sensory impulse passing up the spinal cord. Even the impulses for spot-naming ultimately cross to the side of the spinal cord opposite to their point of entry.

But, with lesions of the cortex and subcortical systems, the loss of appreciation of passive position and movement is found on the opposite side to the lesion. It is obvious, therefore, that all afferent impulses, which reach consciousness and are manifested as a disturbance of sensation, ultimately cross to that half of the nervous system opposed to their point of entry. Some cross quickly; but with some, like those which underlie the sense of passive position and movement, the crossing is delayed until the spinal cord has passed into the medulla oblongata and pons varolii.

Now, sensory impulses which pass up the same side of the spinal cord have not reached the second level of the sensory nervous system. For the most distinctive feature of the impulses at a secondary sensory level is the fact, that the remote results caused by interference with them is manifested on the opposite side of the body.

Thus, it is evident that the impulses underlying the

appreciation of passive position and movement and tactile discrimination have not attained, within the spinal cord, to the chief characteristic of sensory impulses at the second or intramedullary level of the nervous system.

Each of these two sets of impulses reaches the spinal cord in combination with others. The fibres of the epicritic system conduct, not only the impulses underlying tactile discrimination, but also those for the appreciation of light touch. The peripheral system, associated with deep sensibility, conducts impulses for pressure, for the pain of excessive pressure, as well as those impulses which underlie the sense of passive position.

Thus, by the time the impulses, which pass up in the epicritic system, have reached the cervical region, those for tactile discrimination have parted company with all the others, which have passed to the opposite side of the spinal cord.

In the same way, of all the sensory impulses which reach the cord by way of the deep system of nerves, those concerned with the sense of passive position and movement alone remain uncrossed. Those associated with the pain of excessive pressure and with the appreciation and localisation of deep touch, have all crossed to the opposite side of the nervous system within the spinal cord.

Among the many sets of impulses poured into the spinal cord by way of the epicritic and deep systems, a gradual filtering off takes place. At last, in the upper cervical region, none from the epicritic system remain uncrossed, except those concerned with tactile discrimination; none from the deep system, but those which underlie the appreciation of passive position and movement.

When we turn to the impulses underlying tactile localisation, we find that, although they have crossed to the opposite side within the spinal cord, they still remain intimately associated with the impulses for tactile sensibility. The epicritic system brings to the spinal cord the impulses for light touch and cutaneous localisation; the deep system carries those concerned with the recognition and localisation of pressure touch (deep touch).

These are immediately transformed and combined into a single group of tactile impulses, which ultimately cross to the opposite half of the spinal cord. Those impulses, associated with tactile localisation, might have been split off to run a course of their own apart from those for tactile sensibility. But this is not the case at the spinal level. Here these two groups of impulses seem to be still intimately associated together. Their fate, at a higher level of the nervous system than that with which we are now concerned, will be the subject of a further communication.

In conclusion, we believe that among the impulses concerned with the local aspect of sensation, which can be interrupted by intramedullary lesions, the three groups do not all belong to the second sensory level. Those concerned with tactile localisation cross the cord and show evidence of recombination; they have, therefore, become sensory impulses of the true intramedullary level. The impulses underlying tactile discrimination and the recognition of passive position and movement show no evidence of recombination, and are interrupted on the same side as motion by intraspinal lesions. From a study of disease higher up the nervous system, above the spinal cord, we know that these impulses ultimately cross, and become recombined. Thus we believe, that a lesion of the spinal cord interrupts these impulses before they have reached the second sensory level. Throughout the spinal cord, they still remain impulses of the primary or peripheral sensory level.

Chapter VII.

General Conclusions.

For fifty years physiologists and clinicians, stimulated by the work of Brown-Séquard, have attempted to trace the upward paths of afferent impulses through the spinal cord. But, in spite of the many admirable experiments on animals and a certain number of well-observed cases of intramedullary disease in man, the latest writers either confess to ignorance, or believe that every impulse passes by at least two paths.

This widespread scepticism arises from the nature of the methods employed. On the one hand, although the experimenter can produce a lesion of the spinal cord in a healthy animal, which can be killed at any desired period after the operation and the secondary ascending degenerations be traced with exactitude, yet it is impossible to say certainly how sensation is affected by the lesion, even when the animal has been long under observation.

On the other hand, the clinician may expend much time and trouble in determining the nature of the loss of sensation; but it is especially in these well-observed cases that it is usually impossible to verify the anatomical nature of the lesion. Most of the instances, in which a careful microscopical examination has been possible, have been cases where the lesion has been progressive, or where the patient has died from complications which greatly detracted from his suitability for psychophysical examination.

No one who has read our paper will think we do not value the experimental or clinical work of our predecessors. If we may seem to pay less detailed attention to it than is customary, it is solely because we have attacked the problem from a different aspect. We accept the experimental result that in animals it is impossible to abolish the upward passage of all afferent impulses from one limb by hemisection of the spinal cord, and fully recognise that the majority of clinical reports are anatomically unsatisfactory.

Recognising the inherent difficulties of both methods, we determined to attack the problem from a different aspect. All previous workers have assumed that afferent impulses reach the spinal cord in the same combinations as those ultimately received by the centres in the brain. Whatever psychical sorting might take place among sensations, it has been assumed that the sensory impulses pass from the periphery to the highest physiological centres, unchanged in quality and in unaltered combinations.

But Head, Rivers and Sherren showed that this could not be the case; for the afferent impulses passed up the peripheral nerves in combinations entirely foreign to those familiar to the investigators of normal sensibility. They

showed that sensory impulses reach the spinal cord by way of the fibres of the posterior nerve-roots in the same remarkable combinations. It was, therefore, certain that the grouping of these impulses must have changed in some way. Where this change occurred, and how the sensory impulses were combined anew, was the problem we set before us.

(*a*) *Results obtained by the Physiological Method.*

Our method has been to trace known sensory impulses, arriving by way of normal peripheral nerves and posterior roots through a diseased spinal cord to higher centres that are entirely unaffected, and we have attempted to indicate how sensation is modified under such conditions.

We showed in Chapter II., how the disturbance of sensation produced by an intramedullary lesion differed from that due to division of peripheral nerves. Impulses arrive by three main streams, along the fibres of the protopathic, epicritic and deep systems. We compared this grouping of impulses at the primary, or peripheral, level with the changes in sensation produced by a purely intramedullary lesion; and for this purpose, we chose the loss of sensibility on the opposite half of the body to that on which motion and reflexes were affected.

All sensory impulses which have crossed the cord show signs of recombination. The form assumed by the loss of sensation shows that the tactile elements have become united into a single group, by whatever peripheral path they may have reached the spinal cord. In the same way, the impulses started by cutaneous painful stimulation arriving by the protopathic fibres, and those due to painful pressure arriving by the deep system of nerves, become combined, parting company with all other sensory impulses that may have travelled with them in peripheral paths. The thermal impulses of the epicritic and protopathic systems become recombined, so that an intramedullary lesion can interrupt all those subserving the sensation of heat, without interfering with the impulses underlying sensations of cold. Or, in some cases, the patient may be insensitive to cold over parts completely sensitive to heat of all degrees. In

passing from the peripheral to the intramedullary, or secondary, level, the impulses change from a regional and developmental grouping to physiologically specific combinations.

Finally, we analysed the loss of sensation caused by intramedullary disease at the level of the lesion, and showed that, in character, it agreed with that remote loss on the side opposed to the lesion where the sensory impulses had been interrupted after they had crossed the spinal cord. Thus, the change must have occurred on the same side of the cord as that by which the sensory impulses entered.

(*b*) *The Nature of the Impulses in the Posterior Columns of the Spinal Cord.*

Tactile, painful and thermal impulses, and those associated with tactile localisation, cross in their passage through the spinal cord, and show evidence of recombination. But the sensory impulses which underlie the recognition of passive position and movement and tactile discrimination do not cross within the limits of the spinal cord. Lesions of the higher centres show that, ultimately, even these impulses cross and are grouped anew; but this change cannot be demonstrated by a pure intramedullary lesion.

Injury and disease of the cervical region of the spinal cord prove that an impaired tactile discrimination and loss of appreciation of passive position and movement may form the only remote loss of sensation on the same side as the disturbance of motion. Thus, although these impulses show no signs of recombination and are still impulses of the lowest sensory level, they are no longer associated with the sensory companions with which they travelled in the peripheral nerves. The impulses underlying tactile discrimination, which reached the spinal cord by way of the epicritic fibres, have parted company with the impulses for light touch, for tactile localisation and for intermediate degrees of heat and cold. The deep system of afferent fibres running with muscular nerves, carried to the spinal cord, not only the impulses associated with the recognition of passive position and movement, but also those for the appreciation and localisation of deep touch and for the pain of exces-

sive pressure. Of epicritic impulses, those for tactile discrimination alone remain uncrossed and uncombined in the cervical spinal cord; similarly, of all those which passed into the spinal cord by way of the deep system of fibres, the impulses underlying the appreciation of passive position and movement alone remain uncrossed and uncombined.

For these impulses we must therefore seek some path in the spinal cord, which is the direct continuation of the posterior roots and which remains uncrossed, until we have passed beyond the limits of the spinal cord. Such a path is found in the long extrinsic tracts of the posterior columns. Fibres from the fifth lumbar and first sacral posterior roots can be shown to degenerate up to the nucleus of the postero-internal column, and degeneration can be traced up the complete length of the postero-external column, after division of the posterior roots supplying the hand. But the number of fibres which can be found degenerated in the upper part of the cervical region is but a small fraction of those which show evidence of destruction, as a consequence of division of any one posterior root. All the way up, the posterior columns are giving off fibres to other parts of the cord.

On tracing the sensory impulses throughout their course in the spinal cord, an exactly analogous filtering off occurs. Painful, thermal, tactile impulses ultimately pass from the point of entry to the opposite side of the spinal cord.

But, before they pass across, they have undergone characteristic recombination. That this change occurs in the same half of the spinal cord by which the impulses enter, is shown by the fact that the local loss of sensation produced by a pure intramedullary lesion closely resembles, in character and grouping, that of the remote sensory disturbance which lies over the opposite half of the body.

The rapidity with which the sensory impulses cross to the opposite side varies greatly. Some, such as those associated with pain, heat and cold, seem to have passed completely to the opposite side in the space of five or six segments (Piltz [22], *vide* also Chap. V, p. 617). Thus, in man, an intramedullary lesion may interfere completely with sensory impulses for pain, heat and cold from the opposite

half of the body, without disturbing any other form of sensation. With tactile impulses, the crossing is evidently less rapid; for it is rare to find a remote disturbance of tactile sensibility. But, ultimately, even tactile impulses pass completely to the opposite side of the cord, and a lesion in its upper part may, if sufficiently severe, produce loss of tactile sensibility over the same parts that are analgesic and thermo-anæsthetic. But, until this crossing is finally completed, it is obvious that two paths will be open for tactile impulses. They will continue to pass up the fibres of the epicritic and deep systems in the posterior column, until the highest point in the spinal cord at which those impulses entering by any one posterior root are received by the secondary systems. But, much nearer the point of entry, some impulses will have become transformed and have crossed to the opposite side. Thus, two paths will be open for tactile impulses, one in the primary system of the posterior columns, the other in the secondary intramedullary system. In the first of these paths, the impulses travel untransformed, whilst as soon as they enter the secondary path, they will have been recombined into a specific group, associated with all forms of tactile sensibility.

For the impulses associated with pain, heat and cold, a primary and a secondary path coexist for a short extent only within the spinal cord; for those associated with all forms of touch, this double path seems to extend over a considerable length. Finally, the impulses associated with passive position and movement and with tactile discrimination do not, within the limits of the spinal cord, reach the point where they are recombined, but continue uncrossed to pass along the fibres of primary afferent systems in the posterior columns. It is not until they reach the posterior column nuclei (nucleus gracilis and nucleus cuneatus) that they pass from a primary to a secondary sensory system. Here, at last, the impulses associated with tactile discrimination and with passive position and movement undergo recombination, and cross to the opposite side of the nervous system.

Epicritic and protopathic impulses, and those associated

with deep sensibility, travel along the fibres of the posterior columns like rubble over a graduated sieve. Stones of small size drop through at once, those that are larger pass on further, and some travel the whole length of the sieve to fall into a heap at the end. So, some impulses cross rapidly, others after a longer course; some do not become sifted off until the cord has terminated above in the nuclei of the posterior columns.

These impulses alone can be completely interrupted by destruction of the posterior columns. We therefore find, that the only definite consequence of destruction of the posterior columns is to produce loss of tactile discrimination [compass-test] and of the sense of passive position and movement on the same side as the lesion.

(*c*) *The Significance of the Delayed Crossing of Sensory Impulses of the Secondary or Intramedullary Level.*

What is the significance of this delayed constitution of the crossed tracts of the secondary or intramedullary level? The impulses which result from all forms of painful stimulation pass to the spinal cord by the protopathic fibres, and by the fibres of the deep system which run with the muscular nerves. The impulses from the skin, arriving by way of the protopathic system, probably become almost at once transformed, and pass into an intramedullary system at the level of their point of entry. It is probable that the fibres of the deep system, carrying impulses produced by painful pressure from the same part of the body, do not enter by the same posterior roots as those carrying the impulses produced by cutaneous painful stimuli. Thus, more than one segment of the cord is required, before all the painful impulses from any one part of the body can be gathered together and recombined.

This is probably the cause of that want of correspondence between the extent of the cutaneous and deep analgesia, produced by the local manifestations of an intramedullary lesion (*vide* p. 595). It also underlies the remarkable condition described on p. 597, where an area of pain and hyperalgesia to the lightest pressure, due to a diseased joint, lay within the limits of total cutaneous analgesia.

Such cases show why a certain extent of the spinal cord must be traversed before the transformed impulses from any part of the body can be gathered up in a path on the opposite side of the spinal cord. Interruption at any point after they have crossed may produce a remote analgesia with a definite upper border, but this border will probably lie several segments below the position of the interrupting lesion.

An even greater extent of the spinal cord must be traversed by the impulses associated with light touch arriving by way of the epicritic fibres, and by those for sensations of pressure conducted along the deep system. For, the extent of the epicritic supply of any one posterior root greatly exceeds that of its protopathic fibres; the overlapping is greater, and the epicritic fibres are less completely segmental in distribution.

The local effects of an intramedullary lesion are produced by interference with the paths of the secondary level before they have crossed the cord, whereas the remote effects are due to interruption of these tracts as they pass up towards the third level after crossing the cord.

Disease of the grey matter as such does not produce loss of sensation, except by interfering with these paths of the secondary intramedullary level as they pass through its substance.

The more nearly segmental the arrangement of a primary sensory system as it enters by the posterior roots, the shorter will be the distance necessary for the complete crossing of the impulses it carries into the spinal cord. Thus impulses for pain and sensations of temperature arising mainly out of highly segmented protopathic impulses have the shortest double path.

(*d*) *The Path of Impulses Subserving the Sense of Passive Position and Movement.*

We have shown that the more nearly a primary sensory system is arranged segmentally as it enters the spinal cord by the posterior roots the more quickly will its impulses cross to the opposite side after recombination.

The fibres which conduct the impulses for passive position and movement are probably arranged on a plan different from that of the remaining sensory segmentation. The greater number start from end-organs in the joints, muscles or tendons of the limbs, and in the leg, chiefly from the foot and ankle. When the sense of passive position and movement is disturbed, the loss of sensation is arranged according to joints.

Thus, we should not expect the crossing of these impulses to occur as rapidly as those subserving thermal and painful sensibility. In fact, we find that complete crossing and recombination does not occur, until after these afferent impulses have reached the posterior column nuclei.

But this delay in crossing and recombination of the impulses subserving the sense of passive position and movement, seems to have another cause. We have investigated those afferent impulses only which reach consciousness; but many afferent impulses pass in by the primary or peripheral systems which never produce sensation.

We wish to emphasise a distinction between the fate of sensory and non-sensory afferent impulses, which has not been insisted upon sufficiently of late years. If a tract is shown to degenerate from below upwards, it probably conducts afferent impulses. These impulses may however never reach consciousness; they may be associated with some higher reflex condition, such as the maintenance of equilibrium, or the tone of muscles. This may be an entirely unconscious process, and sensation may never be evoked, until some disturbance of equilibrium produces impulses which, passing up sensory paths, reach the highest centres concerned with sensation. A disturbance of non-sensory afferent impulses produces an abnormal condition in the organism. This abnormal condition evokes fresh afferent impulses which, passing up sensory paths, causes the organism to become aware of the change that has occurred in a state normally outside consciousness.

Now, the afferent impulses, passing by means of the deep system from tendons and joints, are pre-eminently associated with the higher reflex states, such as equilibrium. Recent

work, especially that of Horsley and of Sherrington, has shown that the cerebellum is the afferent centre for these higher reflexes. Any column, therefore, which conducts impulses from the tendons and joints will probably give off collaterals to afferent cerebellar tracts.

One such path, the direct cerebellar tract, lies on the same side as the posterior roots by which the impulses from the deep system enter the spinal cord.

It is therefore probable that, in their passage through the spinal cord, the afferent impulses of deep sensibility, concerned with the position and movement of joints, divide into two groups—those destined after transformation to become the non-sensory afferent impulses of the direct cerebellar tract, and those which continue up the fibres of the primary system in the posterior columns to subserve the sense of passive position and movement. The latter impulses do not become recombined until they reach the nucleus gracilis and nucleus cuneatus; they then cross rapidly to the opposite side like every other sensory impulse.

(*e*) *The Theory of Intramedullary Specific Receptors.*

All sensory impulses are ultimately recombined, on passing from the peripheral to the intramedullary level. This recombination takes place in the same half of the nervous system as that by which the sensory impulses enter. Impulses from the primary level which have been initiated by painful stimuli of whatever kind, are united into a single group. In the same way, all tactile impulses, whether they arrive by way of the epicritic or deep systems, are gathered up and travel together in the secondary system.

We believe this is due to an arrangement somewhat as follows; each end-organ in the skin is capable of reacting to the mass stimulations of the environment in a specific manner; hot spots react to certain degrees of heat only, cold spots to cold or, paradoxically, to heat above 45° C. The end-organs of the deep system react to pressure, whether it be painful or not, but do not respond to stimulation with heat and cold.

When these peculiar impulses reach the spinal cord, they are discharged into the secondary systems, each of which is guarded by specific receptors. These act towards the impulses of the primary sensory level exactly as the end-organs in the skin acted towards the mass-stimuli of the environment. When an impulse, which has originated in the effective stimulation of a heat spot belonging to the protopathic system, reaches an intramedullary receptor of the secondary system set aside for the impulses of heat, it starts a specific impulse. But the same receptor reacts to the epicritic impulses which are started by stimulation of the skin with temperatures between 34° and 40° C. No reaction, however, occurs if the receptor of the secondary system is exposed to impulses due to any other form of stimulation.

Similarly, the receptors of the secondary system arranged for painful impulses respond to all impulses of this nature, whether they arise from stimulation by a prick, or from excessive pressure.

In the same way, all tactile impulses are gathered up by the receptors of the secondary tactile system, whether they are started by a light touch and arrive by way of epicritic fibres, or by pressure and reach the spinal cord through deep afferent fibres running with muscular nerves.

Thus, we imagine that, at the point where sensory impulses pass from the primary to the secondary level, there is a mechanism capable of responding to one specific sensory impulse, however that impulse may have been originated.

It is as if the gallery of a concert hall were fitted with a series of resonators, each of which was tuned to a certain note. Each resonator would pick up a peculiar tone, whether it was produced by the strings, the brass, or the wood-wind.

(*f*) *Sensory Impulses of the Secondary Level undergo further recombination at the Third Sensory Level.*

When sensory impulses pass from the primary or peripheral to the secondary or intramedullary level, they are

were firm in their assertions that the sensation was not painful; and yet, an observer watching their behaviour would suppose they were undergoing intolerable pain. This reaction was not evoked on the analgesic side by a purely painful stimulus such as the prick of a pin.

Evidently, stimulation with a strong interrupted current normally causes pain, which rising to intolerable limits obscures all other sensations. But, if the painful impulses are interrupted by an intramedullary lesion, the tactile side of this stimulus is revealed, and, when the current is strengthened, can produce a sensation painless, but intensely disagreeable.

Thus, at the second, or intramedullary level, even the impulses of tactile sensibility can subserve a discomforting sensation.

(*g*) *Conclusion.*

In conclusion, we believe that the spinal cord is the seat of the transmutation of most of the impulses of the peripheral into those of the secondary level of the afferent nervous system.

This transmutation and recombination takes place on the same side as that by which the impulses enter the spinal cord. The secondary paths for sensory impulses then cross with greater or less rapidity, so that ultimately all except those subserving the sense of passive position and movement and tactile discrimination have passed to the opposite side within the limits of the spinal cord. Even these sensory impulses cross after reaching the nuclei of the posterior columns.

At the same time, within the spinal cord afferent impulses become separated into sensory and non-sensory. Of the latter, many pass up in the secondary system of the direct cerebellar tract to reach the cerebellum.

Thus, the mechanisms of the secondary or intramedullary level are concerned with the separation of non-sensory from sensory afferent impulses, and with the recombination and transmutation of sensory impulses into specific groups.

recombined in such a way that those which have been initiated by painful, thermal or tactile stimuli are brought together into specific groups. Interruption of sensory paths within the spinal cord produces a loss of sensation so nearly specific in character, that it might be thought no further recombination was possible.

Closer examination of the groups of sensory impulses at this level shows that they have still to undergo further sifting; the nature of this change in grouping at the tertiary level will form the theme of a further communication. It must be remembered, that intramedullary tactile impulses are still closely associated with those of tactile localisation, and no case has come under our notice where the patient was unable to name the spot touched, unless at the same time tactile sensibility was distinctly diminished. This is a relic of the close relation of sensibility to light touch with cutaneous localisation, and of sensibility to pressure with the localisation of deep touch. Finally, at the tertiary level, all the impulses subserving localisation are brought together, and the impulses for tactile localisation become separated from those for contact sensibility.

Moreover, at the second or intramedullary level, specific sensory impulses carry with them the factors which determine the feeling-tone of the sensation they ultimately subserve. When a strong interrupted current is applied to the foot, the patient cries out with pain and withdraws the leg. Suppose, however, that the lower extremity is totally insensitive to painful stimulation of all kinds, in consequence of an intramedullary lesion, the current, even when of excessive strength, cannot cause pain. But so long as tactile sensibility remains perfect, he will complain bitterly of the discomfort caused by this form of stimulation.

Careful experiments with G. G. (No 3, p. 656), and W. M. (No. 5, p. 667), both of whom were unusually trustworthy and willing patients, showed that the movement of withdrawal seemed to be almost as violent when a current of known strength was applied to the analgesic as to the normal leg. G. G. said the sensation produced was a "kind of exaggerated tickling more unpleasant than pain." Both these patients

leg. Pieces of coal falling upon it caused him no pain, and he was often unaware that he had been struck, until he saw bruises on the leg at night. Further, he found that the leg was insensitive to pinching. He did not trouble himself about this loss of sensation, but on August 13, 1905, he began to have pains in his joints, and was admitted to the London Hospital, on August 16, with an attack of acute rheumatism.

When he was 20 years old, and again at the age of 31, he had had attacks of rheumatic fever, and, for the last twelve months, had been a little short of breath on exertion. He denied syphilis and gonorrhœa. He married at 21 years old, had one child, aged 2 years, who is quite healthy. His wife had previously had three miscarriages.

His work was heavy, as he had not only to drive the van, but also to load and unload the coals. He drank about ten pints of beer daily, but no spirits, and smoked about an ounce of shag a day.

There was no family history of nervous or rheumatic disease. He was liable to urticarial wheals, and factitious urticaria could be produced by pricking or scratching the skin all over the body, both over anæsthetic and over normal parts.

The day after admission, his temperature was 102° F. [38·8° C.], and he had a good deal of pain in the left knee, the left ankle and in both shoulders. He also experienced an uncomfortable sensation in the right knee, and he objected to flexion of the joint, but stated that the sensation was not at all painful; it resembled an itching and was very disagreeable.

The temperature and pain rapidly subsided under the influence of sodium salicylate.

The heart was somewhat enlarged and showed the signs of mitral regurgitation of moderate severity. Compensation was well maintained.

Since this date, the patient has been under frequent observation, and in the following account we shall summarise his present condition.

He is a tall, spare, but very muscular and powerful man, intelligent and answering readily to the various forms of stimulation. His speech is normal, and he has never suffered from fits.

Motion.—The grasp of the left hand was a little weaker than that of the right, but this difference was much increased during the attack of rheumatism, when the dynamometer readings were:—

Right grasp 30 kilograms. Left grasp 7 kilograms.

APPENDIX.

Reports of Cases quoted in the Text.

CASE 1.—FREDERICK C.

He was suddenly attacked with weakness of the legs and left arm, December 13, 1903.

On Jan. 4, 1904, *he showed weakness of left arm and disturbed sensibility over the right half of the body and right leg.*

Seen by us August, 1905. *Well compensated mitral regurgitation.*

Wasting of small muscles of left hand.

Knee-jerks brisk, no ankle-clonus, left plantar reflex gave a flexor response.

Total insensibility to tactile, painful and thermal stimuli was present over the right leg and right half of the trunk (*fig.* 40).

Frederick C., aged 37, a coal porter, went to bed perfectly well on the night of December 13, 1903. When he tried to get out of bed at six o'clock next morning, he found that he could not use his legs. He fell to the ground and had to be helped back to bed. The same afternoon he discovered that his left arm was useless. He was nursed at home for a fortnight, and the weakness of the legs disappeared; but he was still unable to use his left arm. On January 4, 1904, he was admitted as an in-patient to the National Hospital, Queen Square, where he remained for five and a half weeks. His condition was then as follows: "There was weakness of the left arm and loss of sensation over the right leg and the right side of the trunk; the condition was one of complete analgesia and thermo-anæsthesia, with relative loss of sensibility to touch over this half of the body."[1]

He improved greatly whilst in the hospital, and returned to work on the Tuesday after Easter, 1904. The weakness of his left arm had disappeared to such an extent, that he was able to carry nine tons of coal on the first day he resumed work. Soon, however, he began to notice that sensation was lost in the right

[1] For this note we are indebted to the Registrar of the National Hospital, Queen Square.

He was examined on many occasions after the rheumatism had passed away, and the average results were as follows : —

Right grasp 30 kilograms. Left grasp 23 kilograms.

There was slight but distinct wasting of the first dorsal interosseous muscle and of the muscles of the thenar eminence of the left hand. All the movements of the hands, forearms and arms

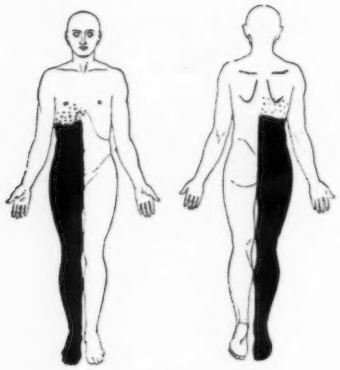


FIG. 40.

To show the loss of sensation to all forms of tactile, painful and thermal stimuli in Case 1. The frontal border was not sharply defined, but merged gradually into parts of normal sensibility.

could be readily performed, and no wasting could be detected in the remaining muscles of either arm. The gait was normal. He did not drag either leg, and it could not be said that one leg was held more stiffly than the other. He could stand steadily with both eyes closed, and even balance on either foot with equal ease. There was no wasting or tremor in either leg. He could approximate his great toes with his eyes closed, even when he had not previously seen the position of his limbs.

The electrical reactions of the muscles in both arms and legs were normal.

Reflexes.—The knee-jerks were brisk, normal and equal, and the plantar-reflex, although difficult to obtain, seemed to give a normal flexor response on both sides.

Sensation.—F. C. stated that the right leg always seemed to him to be hot, as if it were before a blazing fire. When he placed the left leg upon it, or when he touched the right leg with either hand, the right leg always seemed to be hotter than the left.

Pain—(*a*) Stimulation by pricking with the point of pin elicited no reply over the whole of the right leg and over the right side of the body as far as the level of the xiphisternum. The upper border was a soft one and shaded gradually into parts of normal sensibility. The anæsthesia was limited exactly to the middle line and included the skin covering the right half of the penis and the right side of the scrotum.

No pain was produced over the right leg by pressure which would have been unendurable over the left leg. The right side of the body as far up as the level of the cutaneous analgesia was also insensitive to the pain of deep pressure. Not only was the pain of deep pressure lost, but the patient also failed entirely to recognise any increase in pressure even when 15 kilograms were used. The algometer records are given below.

ALGOMETER.

	R.		L.
Sole	15 [no pain]	..	5 [pain].
Shin	13 [no pain]	..	2·5 [pain].
Outer side of knee	20 [no pain]	..	4·5 [pain].
Front of thigh	15 [no pain]	..	5 [pain].
Outer surface of the ant. sup. iliac spine ..	15 [no pain]	..	3·5 [pain].
Lower ribs within the limit of the analgesia..	10 [no pain]	..	1·5 [pain].
Sterno-clavicular joint	3 [pain]	..	3 [pain].
Temple	2 [pain]	..	2 [pain].
Elbow	4·5 [pain]	..	4·5 [pain].

He was insensitive to the interrupted current in all its forms over the whole of the analgesic half of the body and over the right leg. He could appreciate neither the pain nor the vibration of the current. But, as soon as the muscles were contracted so as to produce movement of a joint he said, "You are doing something to me." This occurred every time that any considerable movement of a muscle was induced.

(*b*) *Thermal stimuli.*—He was insensitive to stimulation by water at 50 C°., or by ice, over the whole of the right leg and right half of the body so far as the level of the xiphisternum. The upper level of the thermo-anæsthesia was not sharply defined, and there was an area of skin extending upwards for about an inch and a half over which sensibility to temperature

was diminished. The thermo-anæsthesia was limited exactly by the middle line. Over this area, stimulation with the intermediate degrees of temperature likewise failed to evoke any response.

(*c*) *Tactile sensibility.*—There was profound loss of sensibility to cotton-wool over the whole of the right leg and right half of the body, the upper limit extending to nearly the same level as that of the analgesia and thermo-anæsthesia.

On testing the two legs with von Frey's hairs, it was found that No. 8 [830 mgm.] was everywhere promptly appreciated and accurately localised over the whole of the left leg. No contact with any of these hairs could be appreciated anywhere over the right leg as far as the upper limit of the anæsthesia to stimulation with cotton-wool.

Pressure steadily applied evoked no response over the whole of the right leg and of the lower half of the trunk on the right side. Over the right thigh and over the lower part of the abdomen on the right side, however, if the instrument by which pressure was made was moved sufficiently violently to displace the muscles, the patient stated that he felt a slight sensation. If the whole of the skin and subcutaneous tissues were picked up between the finger and thumb and violently pinched, he never on any occasion gave any response. The sensation experienced by the patient seemed to depend on the movement of the muscles.

(*d*) *Tactile localisation.*—Localisation of touch was perfect over the whole of the left leg, whether the patient was asked to name the point touched with his eyes closed, or whether he was allowed to grope for the point of contact. The maximum final error was in all cases under 2 cm.

(*e*) *Compasses.*—Over the right leg it was impossible to apply this test, owing to the fact that the patient did not respond at all when touched with the compasses, in consequence of the loss of tactile sensibility.

Over the left leg the results were as follows :—

Outer side of left leg (longitudinal).	10 cm.	1	II.	I.	II.	IIIII.
		2	II.	II.	IXI.	XIX.
	12 cm.	1	IIII.	I.	I.	II.	II.
		2	IIII.	I.	III.	II.

The answers were given promptly and with decision. On the right half of the abdomen a similar difficulty was encountered, since the patient did not respond at all over this area when

touched. Over the left half of the abdomen the result was as follows:—

Left half of abdomen (longitudinal).	6 cm.	1	III.	II.	III.	II.
		2	II.	III.	II.	III.

On the hands compass sensibility was normal.

(*f*) *Sense of passive position and movement.*—Although there was profound loss of cutaneous and of tactile sensibility in the right leg, the patient was able to recognise any position into which his limb was passively placed. The results obtained were as follows:—

		Right.				
Great Toe	Extension	I.	II.	II.	III.	II.
	Flexion	II.	I.	I.	III.	III.

		Left.				
Great Toe	Extension	I.	II.	III.	III.	I.
	Flexion	II.	I.	I.	III.	III.

There is a little stiffening of the great toe joint which prevents the answers being as good when the toe is extended as when it is flexed.

All movements at the ankles and knees were appreciated with equal accuracy.

Although the patient could not appreciate firm, steadily-applied pressure, he experienced a sensation if his muscles were seized and moved about. If the muscles were made to contract by an electric current, he appreciated the fact that they were being moved. Moreover, he was able to co-ordinate the movements of the muscles of the right leg quite as well as those of the opposite sound limb.

(*g*) *Acuæsthesia and sense of size.*—As might have been expected from the total loss of painful and tactile sensibility, the patient was quite unable to distinguish the head from the point of a pin, or to discriminate between discs of various sizes when applied to the skin of the right leg.

(*h*) *Tuning-fork.*—All sensation of vibration was lost over the right leg, the right iliac crest, and the right half of the abdomen. The vibrations were, however, appreciated over the lower ribs and the costal margin on the right side.

Cranial nerves.—The pupils were equal and reacted well to light and accommodation. No cranial nerve was affected.

Sphincters.—There was no abnormality in micturition or defæcation.

Spine.—No deformity.

Case 2.—Charles B.

Fracture of the neural arch of the third cervical vertebra from a fall on to the head.

Loss of sensation to pain and temperature over the left half of the body. (Fig. 41.)

Sensation to all forms of touch undisturbed.

Sense of passive position and movement, tactile discrimination and tactile localisation perfect.

Laminectomy, June 25, 1905.

After the operation the condition of sensation was unaltered, except that all sense of passive position and movement was lost in the right arm and leg.

On April 11, 1904, Charles B., a man of 59, was thrown out of a dog-cart on to his head. He thinks he lost consciousness, but is certain that such loss was momentary only, for he remembers lying in the road, watching all that was going on around him, completely paralysed down the right side. He was carried to bed and was able to move his toes upon the tenth day after the accident. Power gradually returned to the right leg, and in two months the right arm was so far recovered that he was able to write.

From the moment of the accident he has been insensitive to heat and cold over the left half of the body. From the left hip downwards he has suffered from a burning sensation, which seems to lie deep below the surface and is always worse after a good meal. He has also suffered greatly from cramp in the right leg.

Condition in May, 1905.

He was a spare, well-built man, looking fully his age. His vessels were not abnormally thickened, and the heart and lungs showed no disease.

Motion.—He walked like a man with hemiplegia, lifting the right hip, and swinging the right foot, so that the toes cleared the ground. He could stand steadily with his eyes closed even when his feet were placed together. As he lay on a couch, there was very little loss of power in the right foot or right knee, when the movements were made against resistance. He could dorsiflex the right foot at the ankle, but the movement was somewhat slower than on the opposite side.

The grasp of the right hand was decidedly weaker than that of the left; all movements at the right wrist and elbow could be

performed, though they were less strong than on the opposite side. He could touch his nose with the forefinger of either hand, even when the eyes were closed, and showed no signs of ataxy. The supraspinatus, the infraspinatus and deltoid muscles were slightly wasted; but it must be remembered that the right shoulder joint was fixed by adhesions. The action of the deltoid was weak, but the biceps and supinator longus contracted strongly; none of the small muscles of the hand were wasted. All the muscles reacted perfectly to the interrupted current, even those that appeared to be wasted.

Reflexes.—The right knee-jerk was greatly exaggerated. Ankle-

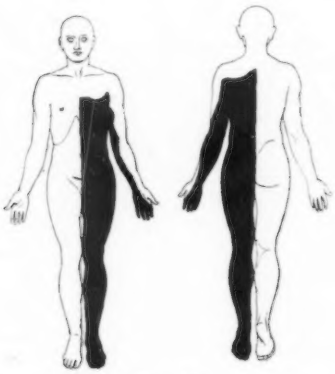


FIG. 41.

To show the loss of sensation to painful and thermal stimuli in Case 2.

clonus was easily obtained from the right foot, and the right toe gave a characteristic extensor response on stimulation of the sole. The right wrist-jerk was considerably in excess of the left. The left knee-jerk was diminished. No ankle-clonus could be obtained on this side, and the plantar reflex gave a definite flexor response.

Sensation.—He complained of a sensation of burning in the buttock down the left leg, principally on its outer side. A similar sensation troubled him in the left arm, mainly situated in the back of the hand.

Over the left half of the body, below the line marked in Fig. 41, all forms of painful stimulation and all degrees of temperature failed to produce any sensation. The border of this abnormal

area seemed to be well-defined on the arm, but towards the periphery of the limb faded into parts of normal sensibility.

The lightest touch with cotton wool produced a sensation in every part of the body, and to this stimulus there was no difference between its two halves. Pressure was everywhere appreciated equally.

Cutaneous localisation was excellent to both light touch and deep pressure, and no difference could be discovered in the accuracy of localisation on the two sides of the body.

He was able to recognise the position into which his limbs had been moved passively, and his answers were quick and accurate, even for movements of the great toes.

The vibrations of the tuning-fork were appreciated everywhere.

Over the left half of the body and left leg, the strongest interrupted current produced no pain; but he said that a strong current gave him exactly the same sensation as that produced by holding a hot plate.

Sphincters. — Micturition and defæcation had never been affected in any way. At first, he was impotent without the power of erection, but in May had regained his power, and emission occurred, though more slowly than usual.

Cranial nerves.—The pupils were equal and reacted normally, dilating well to shade. The optic disc showed no hyperæmia and the fundus was normal. The face and tongue were not affected.

Spine.—The spine of the second cervical vertebræ had sunk in consequence of collapse of the third cervical, and a radiograph showed this condition distinctly.

Subsequent Progress.

On June 6, 1905, he was seen by Sir Victor Horsley, who admitted him to the National Hospital, Queen Square.

On June 25 the laminæ of the second, third, fourth and fifth cervical vertebræ were freely exposed; the third neural arch was found to have been fractured and pushed downwards. There was, however, no evidence of pressure on the spinal cord. The third arch was removed and the probe was passed both upwards and downwards, without the discovery of further fracture or pressure on the cord. But, in order to make certain, the laminæ of the fourth vertebræ were cut through. The cord was seen to be pulsating well and there was no sign of hæmorrhage within the membranes. The wound was then closed. The patient stood the operation unusually well and showed little evidence of shock.

He recovered perfectly from the operation and the wound

healed by first intention. For the first fortnight he was completely free from pain or abnormal sensations, but, at the end of that time, the burning in the left side returned worse than before. At the same time he noticed that the right arm seemed stiff and cramped.

In October, 1905, he was again seen by one of us. He complained that "his right leg felt exactly as if it was a cork leg," and that all the muscles on this side "felt tight." He believed that he had lost power both in the right arm and leg since the operation.

Motion.—He still walked with a hemiplegic gait, and there was no obvious increase in the loss of muscular power. But all movements in the right leg and right arm had become more difficult in consequence of definite ataxy. He could no longer touch the tip of his nose with his right forefinger, nor bring the two forefingers together when the eyes were closed. He could not stand on his right foot with any certainty, and on closing his eyes fell at once.

There was no further wasting beyond that noticed in May.

Sensation.—Over the left half of the body and left leg all sensation of pain, of heat, and of cold was abolished as before. (Fig. 41.) Light touch and pressure were perfectly appreciated and localised with certainty. The sense of passive position and movement was excellent, and the compass-test gave normal results; the vibration of the tuning-fork could be everywhere recognised.

Over the right leg, the right half of the body and the right arm, cutaneous painful stimuli were normally appreciated. Sensations of heat and cold were perfect. Sensibility to light touch and pressure was equal to that on the opposite side; but a remarkable change had occurred in the pain produced by increasing this stimulus. All down the right side and in the right leg, pressure with the head of a pin produced a painful sensation. There was no tenderness when the point was dragged gently across the skin, or when it was picked up between the fingers. But, as soon as the head of a pin was lightly pressed anywhere over the right half of the body, he complained of pain. Deep tenderness was certainly present.

Moreover, he was no longer conscious of the position into which his thumb, index and middle fingers of his right hand were placed passively. He also had considerable difficulty in recognising the position of his wrist. No passive movement of the toes of the right foot could be recognised, and his answers were frequently inaccurate after movements of the right ankle. None of the other physical signs were in any way altered.

Shortly afterwards he left England, and we have had no further opportunity of examining him.

Case 3.—Grace G.

Sudden loss of power in the right leg on February 18, 1898, whilst sleeping.

The condition of motion and sensation has shown no material change since August, 1898, when she first came under our observation.

The right leg is spastic and paretic. The right knee-jerk is much exaggerated, ankle-clonus is obtained on the right side and the plantar reflex gives an extensor response. On the left side the reflexes are normal, no ankle-clonus is present and the plantar reflex gives a flexor response.

Complete loss of sensation to pain and temperature over the left lower extremity (Fig. 42).

All forms of tactile sensibility are perfect.

Sense of passive position and movement and tactile discrimination (compass-test) are disturbed in the right foot.

No other abnormal signs are present in the nervous system.

Grace G., a married woman, aged 39, lay down to sleep as usual at 2 p.m. on the afternoon of February 18, 1898; she was then in perfect health. On rising an hour later, she was seized with pains round the body at the level of the umbilicus. She cried out for help and was found on her feet, holding the edge of the bed. She did not fall, but, when helped into bed, discovered she had lost all power in the right leg. For the first seven days she suffered with pain down the outer side of the right leg, which remained absolutely paralysed for three weeks. At no time was there any loss of power in the left leg.

From the first, she was able to recognise when the paralysed right leg was pricked, but it was not until four weeks after the onset that she discovered, on taking a bath, she was unable to discriminate heat from cold when applied to the left leg. The paralysed limb has remained throughout sensitive to these stimuli.

The complete paralysis of the right leg passed away gradually within the first two months; the leg became stiff and has remained in the same condition until the present time. For eight years the sensory condition of the left leg has remained unaltered.

The date of the attack coincided with the first day of a normal menstrual period. She is usually regular at intervals of twenty-eight days; the menstrual flow lasts for five days and is not usually profuse. Occasionally, however, since her marriage, she has gone two to three weeks over her normal time, and then the flow has been more profuse than usual. Five months after her marriage, she had an attack of what was thought to be peritonitis; she suffered much pain and was in bed for three weeks. During this time there was a constant discharge of blood from the vagina. This was possibly a miscarriage; but at no time has she ever been certain that she was pregnant.

There is no history of venereal disease and nothing pointing to the probability of infection.

She first came under the observation of one of us in August, 1898, five months after the onset of the paralysis. During the last eight years, she has been repeatedly examined; and, although we have amplified the scope of our observations, her condition has remained unchanged. The observations made in 1898 are in complete accord with those of the present time.

Present Condition.

She is a tall, well-built woman, of unusual intelligence, not anæmic nor wasted, and, except for the difficulty in walking, she says she is in perfect health.

Motion.—She walks with a stick, dragging the right leg, which she holds stiffly. She says she dare not let the right knee go slack, or she would fall. This leg is rigid and spastic; but the rigidity can be overcome by the exercise of steady pressure. At night she suffers from involuntary "jumping" of this leg. The right foot is in a position of slight pes cavus, due apparently to the extreme extensor response to all stimulation of the sole.

It is difficult to test the amount of voluntary power in this leg on account of its spasticity. But both dorsiflexion and plantar extension are feebly performed at the ankle-joint and flexion of the knee and hip is weak. Extension of the knee and hip can be more perfectly carried out, though less strongly, than with the left leg.

There is no profound wasting of either leg, but the right calf is one inch (2·5 cm.) smaller than the left at its largest circumference.

The motor power of the left leg is unaltered, and the upper extremities are entirely unaffected.

The electrical reactions, even of the right leg, are everywhere normal.

Reflexes.—The right knee-jerk is greatly exaggerated and ankle-clonus is easily obtained. The right great toe gives an extreme extensor response when the sole of the foot is stimulated.

On the left side the knee-jerk is brisk, but there is no ankle-clonus. The great toe either does not respond or occasionally gives a faint flexor response.

Sensation.—She complains that the right leg nearly always seems to her to be cold; but the left leg is never hot nor is it

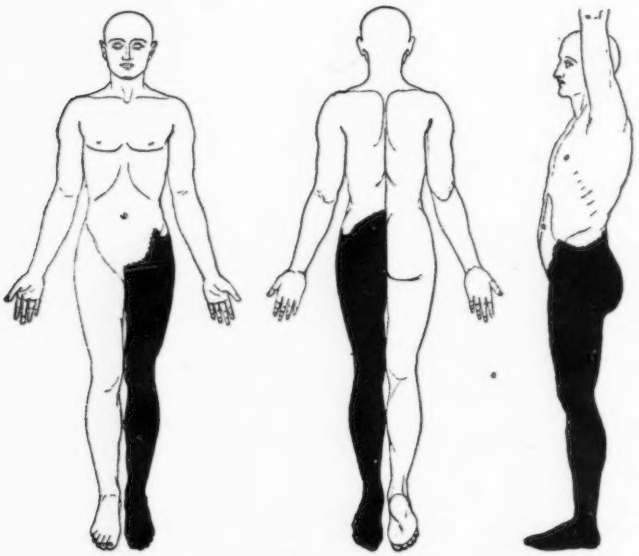


FIG. 42.

To show the loss of sensibility to painful and thermal stimulation in Case 3.

The border between the normal parts and those which were completely insensitive was unusually well defined, corresponding to the caudal border of the eleventh thoracic segment.

cold even in the winter. When she touches the right leg it always appears to be colder than the left. But, apart from these symptoms, she is not troubled by any pain or other abnormal sensation.

(*a*) *Loss of Painful Sensibility.*—The prick of a pin caused no pain over the whole of the left leg and thigh as high as the

level of the iliac crest. The boundary between sensitive and insensitive parts was clearly defined, following the line marked upon Fig. 42. So sharp was this border that it made little or no difference whether the observations were made from the direction of normal to abnormal skin or *vice versâ*.

The algometer gave the following readings, showing that the parts insensitive to a prick were also insensitive to the pain of pressure.

	R. [motor loss].	L. [analgesic and thermo-anæsthetic].
Sterno-clavicular joint	3 [pain] ..	3 [pain].
Palms	4 [pain] ..	4 [pain].
Lower ribs	2 [pain] ..	2·5 [pain].
Outer aspect of ileum close to ant. sup. spine	3·5 [pain] ..	10 [pain].
Front of thigh ..	4 [pain] ..	14 [no pain].
Inner aspect of knee	5 [pain] ..	15 [no pain].
Shin	2 [pain] ..	11 [slightly uncomfortable].
Sole of foot	1·6 [pain] ..	12[1] [uncomfortable].

The loss of sensation to the painful interrupted current corresponded exactly to the area insensitive to prick. But she complains that when the current is applied to the left leg, which is completely analgesic, an exaggerated tickling sensation is produced. This is disagreeable and is associated with rapid withdrawal of the leg.

(*b*) *Heat and cold.*—Over the whole of the area on the left leg, shown in Fig. 42, she was entirely insensitive to all degrees of temperature. Over the right leg all temperature stimuli were correctly appreciated.

(*c*) *Tactile sensibility.*—Stimulation with cotton-wool produced a normal sensation, which was in no way different in the two legs. Pressure was everywhere correctly recognised, and even over parts where the algometer produced no pain the increasing pressure was distinctly appreciated. When tested with von Frey's hairs, no difference could be discovered between the two legs, but she was somewhat insensitive to this form of stimulation over both lower extremities. No. 5 [360 mgm.] was as frequently appreciated over the left as over the right leg; but over both legs she not infrequently failed to recognise this stimulus.

(*e*) *Acuæsthesia and appreciation of differences in size.*—Over

[1] She complains of an uncomfortable tickling when the algometer is pressed on to the sole of the left foot, which greatly disturbs the readings. Sometimes this condition makes it impossible to test the sole of the left foot.

the whole of the analgesic leg she was able to tell the head from the point of a pin; in twenty stimulations to the sole of each foot, ten of which were made with the head and ten with the point, she did not give a single incorrect answer. Her power of recognising the relative size of objects was tested with three metal cylinders, 2 cm., ·75 cm., and ·2 cm. in diameter. In spite of the most varying combinations, her answers were always correct over both legs.

(*f*) *Tactile localisation.*—Light touches and deep pressure were localised with remarkable accuracy, whatever method we adopted. Sometimes she was asked to name the point touched, at others she was allowed to point to the spot with a cardboard-rod. At other times, we adopted the method of touching the skin through a hole in a piece of paper, held a short distance above the skin, and she attempted to mark the spot touched with a soft pencil. At no time were we able to find the least difference between the two legs, and, when allowed to point, her accuracy and quickness was remarkable, although her eyes remained closed.

(*g*) *Recognition of passive position.*—At every joint in the *left* lower extremity (analgesic and thermo-anæsthetic) her answers were remarkably quick and accurate. The position into which the *right* knee and ankle had been placed was also well recognised, but she was greatly at fault with regard to the position of the great toe on the right side. A comparison of the records of twenty stimuli in each foot, arranged graphically, shows this difference in a very striking manner, even the answers that were correct in the right foot were more slowly given than on the left side.

L. [analgesic and thermo-anæsthetic].

Great Toe	Flexion	I.	III.	II.	II.	II.	
	Extension	II.		II.	I.	III.	II.

R. [spastic].

Great Toe	Flexion	II.	X.	IIIX.			III.
	Extension	XX.	I.	XI.		XXIXX.	

L.

Ankle	Flexion	I.	III.	I.	III.	II.	
	Extension	II.		III.	I.	IIII.	

R.

Ankle	Flexion	III.	I.	I.	III.	II.	
	Extension		I.	III.	II.	III.	I.

(*h*) *Compass-test.*—No difference between the two legs could be discovered until the foot was reached. Even over the feet the records, as she lay in bed, were almost equally good on the two sides.

	L. [analgesic and thermo-anæsthetic].		R. [spastic].	
Sole of foot . .	2 cm.	1 | 7 R. 3 W. 2 | 10 R.	2 cm.	1 | 9 R. 1 W. 2 | 2 R. 8 W.
	2·5 cm.	1 | 9 R. 1 W. 2 | 10 R.	2·5 cm.	1 | 8 R. 2 W. 2 | 8 R. 2 W.
	3 cm.	1 | 10 R. 2 | 10 R.	3 cm.	1 | 10 R. 2 | 10 R.
Dorsum of foot	2·5 cm.	1 | 7 R. 3 W. 2 | 10 R.	2·5 cm.	1 | 7 R. 3 W. 2 | 5 R. 5 W.
	3 cm.	1 | 7 R. 3 W. 2 | 10 R.	3 cm.	1 | 7 R. 3 W. 2 | 9 R. 1 W.

Thus after a rest of two hours in bed there was no material difference between the formulæ at 3 cm.

She then rose and walked 150 metres up and down a corridor. After returning to bed the difference between the two feet were distinct even at 4 cm.

	L.		R.	
4	cm.	1 | 10 R. 2 | 10 R.	4 cm.	1 | 6 R. 4 W. 2 | 6 R. 4 W.

Thus, the only interference with her power of discriminating two points occurred over the right foot, *i.e.*, on the side of the motor paralysis and on the side opposed to the analgesia.

(*i*) *Tuning-fork.*—The vibrations of a tuning-fork were everywhere recognised, and there was no difference between the two legs.

Sphincters.—She has no trouble in holding or passing her urine or fæces.

Cranial nerves.—The pupils react well: disc and fundus are normal. Hearing, smell and taste are perfect.

No other part of the nervous system is in any way affected.

There is no disease of the heart or lungs. The pulse is good, and the vessels show no abnormality.

Case 4.—Albert H.

Fell into the hold of a ship on December 3, 1901.

Is said at first to have been able to move his right leg only, but when first seen by Mr. Sherren and one of us in February, 1902, the loss of power was confined to the left arm and leg.

He is a characteristic instance of Brown-Séquard paralysis.

Right.	*Left.*
No motor disturbance.	*Paresis and wasting of left hand.*
Knee-jerks normal.	*Paralysis of Triceps.*
No ankle clonus.	*Spasticity of left leg.*
Plantar reflex flexor in type.	*Knee-jerks greatly exaggerated.*
Loss of sensation to painful and thermal stimuli over an area shown in Fig. 43.	*Plantar reflex extensor in type.*
Diminution in tactile sensibility over the right foot, with coincident uncertainty in answers to the compass-test.	*Sensation perfect.*
All other forms of sensation perfect.	

Albert H., aged 25, a boiler maker, fell into the hold of a ship on December 3, 1901.

He went below to screw up some frames but did not trouble to take a light. Expecting to step on to the lower deck, he walked into space, and was picked up head downwards unconscious in the hold.

He was taken to Poplar Hospital, where he lay unconscious until the evening.

When he woke he found he could move the right leg only.

In about three weeks he began to recover movement in the right arm. Gradually power came back in the left leg, so that by the end of January, 1902, he could walk; and when seen by Mr. Sherren and one of us in February, 1902, he said power was returning to his left arm.

It is impossible to make any statement with regard to the condition of sensation after the accident, as he confuses sensory with motor paralysis. The dominant facts in his mind concern the paralysis, first of both arms and the left leg, later of the left arm and left leg, and finally the condition which he calls paralysis of the left arm only.

When he first came under the notice of Mr. Sherren and one of us in February, 1902, two months after the accident, his condition was as follows :—

He walked like a hemiplegic, dragging the left foot. He could not grasp with the left hand, and the movements of the left wrist were weak. The intrinsic muscles of the left hand were wasted. Dorsiflexion and plantar extension of the left ankle were feeble, and the left leg was spastic.

The left knee-jerk was greatly exaggerated ; ankle clonus was obtained on the left side, and the left plantar reflex gave an extensor response. The wrist-jerk and elbow-jerk were exaggerated on the left side. All the reflexes in the right arm and leg were normal.

The sphincters were not affected, and there was no alteration in the reaction of the pupils to light or to shade. The left palpebral fissure was smaller and the left ear colder than on the right side.

The whole of the right half of the body below the line on Fig. 43 was insensitive to pain, heat, and cold ; as the condition of sensation has changed little in the last five years, we shall give the further details later.

By July, 1902, he had considerably improved, and could walk without a stick.

Motor power steadily increased until the end of 1902. Since that time his condition has remained stationary. He now keeps a small sweet-shop, and feels in perfect health except for the difficulty in walking.

During the last five years he has been examined repeatedly, and the following account, based on his condition during the latter half of 1906, includes some of the observations made throughout the previous period.

Present Condition.

He is a short, somewhat youthful-looking man of moderate intelligence. He has never been intemperate, and does not now take any alcohol.

Except for the defects in the nervous system he shows no symptoms or signs of disease.

Motion.—He walks like a man with hemiplegia, sweeping the left leg outwards to enable the left foot to clear the ground. At the same time the left leg is kept rigidly extended. He can stand steadily with the eyes open or shut, but has difficulty in standing on either leg alone.

The left leg is distinctly spastic. He can slightly extend the toes but cannot dorsiflex the ankle; flexion of the toes and plantar extension of the ankle are slowly and feebly performed against resistance. If given time he can flex and extend the knee; but the movement is feeble against resistence. Movements at the hip are better performed, and, though slow, are powerful.

The left leg is not wasted.

The left hand is held with the wrist a little flexed and the fingers doubled into the palm. The fingers cannot be extended unless the wrist is fully flexed, in consequence of the shortening of the flexor muscles. He seems to have lost the power of straightening the fingers voluntarily, but can grasp feebly. He

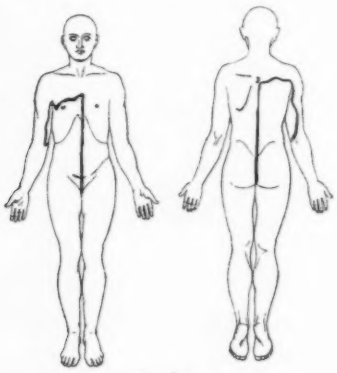


FIG. 43.

To show the border at which sensation to pain, heat and cold ceased in Case 4.

can extend the wrist well until this movement is checked by the shortening of the flexor muscles, and can pronate and supinate the forearm.

The triceps acts extremely badly, if at all; when the arm is flexed and the patient is told to extend it he moves the whole arm until it is jerked into the extended position. The biceps, deltoid and supinator longus contract well, and the muscles of the left shoulder are unaffected.

The interosseous spaces of the left hand are hollowed and all its small muscles are somewhat wasted. The left forearm is 1 cm. and the left arm 1·5 cm. smaller in circumference than the right.

All the muscles of the left upper extremity, with the exception of the triceps, react normally to electrical stimulation. The triceps

reacts badly, if at all, to the strongest interrupted current, and, on closing the constant current, contracts more easily to the anode than to the kathode.

On the right half of the body every motor function has been perfectly normal since he first came under observation, three months after the accident.

Reflexes.—The left knee-jerk is greatly increased; ankle clonus is obtained on the left side, and the plantar reflex gives an extensor response.

On the right side the knee-jerk is normal, no ankle clonus can be obtained and the plantar reflex is of the flexor type.

Wrist and elbow jerks are equal on the two sides.

Sensation.—He complains of no abnormal sensation and says that if he had not been tested he would not have known that sensation was in any way defective.

(*a*) *Sensibility to painful stimuli.*—At the line marked on Fig. 43 a profound change occurs in his sensibility to painful cutaneous stimuli, such as a prick or the painful interrupted current. All sensibility to pain is lost as soon as this line is passed, when travelling from normal to abnormal parts; at the same time, when passing from the analgesic area he becomes sensitive at this line. Throughout the five years he has been under observation this border has always been well defined, and Fig. 43 exactly corresponds with a photograph taken in November, 1902.

He is insensitive to the pain of deep pressure over the right half of the body as high as the lower ribs, and the following readings in kilograms were obtained by means of the pressure algometer.

	R.	L.
Sole	15 (no pain)	4 (pain).
nternal malleolus	12 (no pain)	3·5 (pain).
Shin	14 (no pain)	3·5 (pain).
Inner aspect of knee	14 (no pain)	5 (pain).
Front of thigh	13 (no pain)	5 (pain).
Outer part of ant. sup. spine of ilium	12 (no pain)	3 (pain).
Lower ribs	4 (pain)	2 (pain).
Palm	7 (pain)	6 (pain).
Shoulder	4 (pain)	4 (pain).
Clavicle	2 (pain)	2 (pain).
Mastoid	2 (pain)	2 (pain).
Temple	1·5 (pain)	1·5 (pain).

He was able to recognise the gradually increasing pressure everywhere over the analgesic area, although it caused no pain.

(*b*) *Sensibility to thermal stimuli.*—Throughout the five years,

he has been entirely insensitive to all thermal stimuli over the right leg and right half of the body below the line on Fig. 43.

He has been carefully tested on many occasions with temperatures of 0° C., 20° C., 30° C., 40° C., and 50° C., and found to be insensitive to all degrees. The patch on the inner aspect of the right arm can be perfectly defined with thermal stimuli.

(*c*) *Tactile sensibility.*—Touches with cotton wool can be appreciated everywhere; but over the sole of the right foot his answers are less certain to this stimulus than over the opposite sole. This difference becomes measurable when he is tested with von Frey's hairs. For over the left sole and left shin he recognised No. 8 (830 mgm.) with ease, whilst over similar parts on the right lower extremity this stimulus produced no sensation. This diminished tactile sensibility appears in the defective compass-records obtained over the right sole.

Sensibility to deep pressure was nowhere appreciably different on the two sides of the body.

(*d*) *Compass-test.*—The records obtained to this test were everywhere perfect and equal on the two sides except those obtained from the right sole. Here his answers were slower and somewhat less perfect, and he recognises that this is due to a diminution of tactile sensibility. He says he cannot "feel the touch so well" over the right foot. We believe that this is an instance where the compass-records are disturbed by defective tactile sensibility rather than by a loss of tactile discrimination.

Over the palm his answers are equally quick and accurate at 1·5 cm. But over the soles we obtained the following results.

Compasses applied transversely :—

	R.	L.
2 cm.	1 | 10 R. 2 | 10 W.	1 | 9 R. 1 W. 2 | 9 R. 1 W.
3 cm.	1 | 8 R. 1 W. 1 no answer. 2 | 9 R. 1 no answer (answers given slowly).	1 | 10 R. 2 | 10 R. (answers quick).

At 4 cm. his answers were equally good over both soles.

When the right sole is tested the accuracy of his answers can be greatly improved by practice. Thus he may fail on four or five occasions to answer at all during a series of twenty stimuli with one or two points separated at a distance of 3 cm. But after further testing of the sole with the points at different distances he sometimes gives a perfect series of answers at 3 cm.

(*e*) *Passive position and movement* can be perfectly appreciated on both sides of the body, and there is no difference in the records

obtained from the great toes, ankles, knees, hips, or any joints of the upper extremities.

(*f*) *Tactile localisation* is equally good on the two halves of the body both to naming and groping.

(*g*) *The vibrations of a tuning-fork* were equally well appreciated everywhere over the two halves of the body.

(*h*) *Appreciation of differences in size.*—Cylindrical rods were used with diameters of 2 cm., 0·6 cm., and 0·2 cm. The difference in size could be appreciated equally well over the two halves of the body except the soles, which could not be tested with certainty.

Cranial nerves.—Taste and smell are unaffected. There has been no limitation of the visual field in either eye, and disc and fundus have always been normal.

The pupils are equal and react normally to light and to shade. The left pupil dilates well to cocaine.

The movements of the face and tongue are normal.

Sphincters. — Micturition and defæcation have never been affected in any way.

Spine.—No gross abnormality is visible in the spine, but a radiograph shows that the fourth neural arch has been fractured.

CASE 5.—WALTER M.

May 18, 1904, *he fell* 15 *feet on to a heap of bricks. Immediate loss of power in the right arm and leg. Shortly afterwards defective sensation was discovered in the left leg.*

Seen by us in 1906.

He was then a characteristic instance of Brown-Séquard paralysis.

Right Side.	*Left Side.*
Wasting of muscles of hand.	*No paralysis or muscular wasting.*
Spasticity of wrist and of leg.	
Knee-jerks increased.	*Reflexes normal.*
Ankle-clonus.	*Loss of sensibility to pain, heat and cold.*
Extensor plantar reflex.	
No change in sensation.	*Tactile sensibility, tactile localisation, tactile discrimination, sense of passive position and movement unaffected.*

Walter M., aged 46, a carpenter and joiner, fell 15 ft. on May 18, 1904. He was working on a scaffold, and, in attempting

to cross an opening, supported himself by a wooden rail; this broke, and he fell on to a rough heap of bricks. He did not lose consciousness, but was carried by his fellow workmen to St. George's Hospital, where he lay three or four weeks.

He was much bruised, especially at the "bottom of the spine." The right arm was weak and, to a less extent, he had also lost power in the right leg; but at no time was he unable to move both the leg and the arm. He was told by the house physician that over the left leg he did not answer correctly to the prick of a pin, and to hot and cold tests. This surprised him, for he was not conscious of any defective sensation in this leg.

On leaving the hospital, he was able to walk unsteadily. Gradually his gait improved, and he could do a little work, but his neck seemed to want support. He suffered no pain, but when he sat up or walked for a time, he felt an uncomfortable sensation in the region of the seventh cervical spine, which occasionally became an ache.

He has had no abnormal sensations in the left leg, but when he takes a hot bath he always tests the water first with his right foot lest he should burn himself. When he dips the left leg into cold water, it produces numbness and not a sensation of cold.

He thinks the strength of the right arm and leg is still improving.

He has never had trouble with micturition or defæcation. Since the accident, he has had no connection with his wife, and, although occasionally he may have an erection, there has been no emission. Sexual desire is evidently slight.

He has been married for nineteen years and his wife has had no miscarriages. No history could be obtained of venereal disease. He is a life-long teetotaler and non-smoker.

Condition in October, 1906.

He was an unusually intelligent man, self-educated to a high degree. Somewhat excitable in manner and speech, he was absolutely trustworthy to control-tests.

Motion.—He walked well, holding the right leg a little stiffly with a slight drag of the right foot. He could stand equally well with his eyes open or shut. The right leg was distinctly spastic, and he complained that it "jumped" at night occasionally. Neither leg showed any signs of wasting, and the measurements were equal on the two sides.

The left hand was in every way normal, but the interossei and hypothenar muscles of the right hand were distinctly wasted,

and in them could be seen fibrillary twitchings. Not only were these muscles wasted, but the fingers were rigid; the proximal phalanges were hyper-extended, and the two distal phalanges slightly flexed. The thenar muscles were in a similar condition, much wasted compared with those of the other hand, and the thumb was adducted and flexed in consequence of the spasticity. Every movement of the right thumb could be carried out, though feebly. The grasp of the right hand was 2 kilograms, that of the left 25 kilograms. All the small muscles of the right hand were less excitable to the interrupted current than those of the left; to the constant current they contracted sluggishly, and more easily on closure to the anode than to the kathode.

All movements of the right wrist could be carried out, but were distinctly spastic. Movements of the elbow were normal and there was no spasticity.

The muscles of the post-axial half of the right forearm were somewhat wasted; the difference between the two forearms at a point 20 cm. above the lower end of the radius was 1·5 cm. (right 22 cm., left 23·5 cm.).

All the muscles of the right forearm and arm reacted well to electrical stimulation.

On quiet respiration the right half of the chest moved distinctly less than the left; this difference became more evident when the patient breathed deeply. Similarly, the movement of the left was greater than that of the right half of the abdomen; when told to contract the muscles of his abdomen, the mid-ventral line was drawn over to the left.

Reflexes.—On the right side, the knee-jerk was greater than that on the left, and ankle-clonus was obtained. The right plantar reflex was invariably of the extensor type, whilst flexion was always obtained from the left foot. The right elbow-jerk was definitely brisker than that of the left side.

Sensation.—(*a*) *Sensibility to pain.*—He was entirely insensitive to the prick of a pin over the whole of the left half of the body as high as the level of the third rib, and over a small area on the inner aspect of the left arm (Fig. 44). The limits of this area were well defined in front and on the arm; they varied little according to whether the patient was tested from analgesic parts towards those of normal sensibility or *vice versâ*. Over the scapula the border differed by about 2 to 3 cm. according to the method of testing employed.

When tested with the painful interrupted current, the analgesia was found to agree exactly with that mapped out by means of the prick of a pin.

But, although he was entirely insensitive to the pain produced by the current, it caused violent movements of withdrawal. He complained that the sensation, though in no way painful, was extremely uncomfortable.

Squeezing the right testicle between the fingers caused the usual sickening sensation; when the left testicle was squeezed he appreciated the pressure, but no pain or discomfort was produced.

The pain of pressure was not appreciated as high as the level of the seventh rib on the left side. The algometer gave the following readings:—

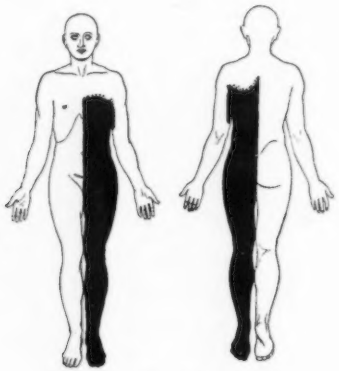


FIG. 44.

To show the loss of sensation to pain, heat and cold in Case 5.

	R. (spastic).	L. (analgesic).
Sole	Pain at 4	No pain at 15.
External malleolus	Pain at 3	No pain at 10.
Shin	Pain at 4·5	No pain at 13.
Outer aspect of knee	Pain at 5	No pain at 12.
Front of thigh	Pain at 5	No pain at 15.
Outer surface of anterior superior iliac spine	Pain at 3	No pain at 12.
Lower ribs	Pain at 1·5	No pain at 10.
Over seventh rib in axilla	Pain at 1·5	Slight pain at 7.
Inner aspect of arm	Pain at 1·5	Pain at 7.
Clavicle	Pain at 1·5	Pain at 1·5.
Palm	Pain at 6	Pain at 6.
Elbow	Pain at 5	Pain at 5.
Shoulder	Pain at 3	Pain at 3.
Temple	Pain at 2	Pain at 2.

(*b*) *Thermal sensibility.*—Any object brought into contact with the area on Fig. 44 was usually said to be warm, even if it were a tube containing ice. This made the testing of thermal sensibility extremely difficult. But, by varying the stimulus and by employing as a control a test-tube at a temperature which seemed neither hot nor cold to the normal parts, we were able to assure ourselves that neither heat nor cold were appreciated over the affected area. If a tube containing ice was passed slowly down the chest from the left clavicle, it was called "warm" as soon as it entered the area on Fig. 44. Similarly, water at 25° and at 45° and the neutral test-tube seemed to him equally "warm."

(*c*) *Sensibility to light touch.*—Stimulation with cotton wool was quickly and accurately appreciated everywhere; no difference could be discovered between the two sides of the body.

Tested with von Frey's hairs we found that No. 5 (360 mgm.) could be appreciated on both palms and both soles. No. 8 (830 mgm.) produced a sensation everywhere on the abdomen, the front of the thighs and the back of both calves.

(*d*) *Deep touch.*—He recognised deep touches and was conscious of the gradual increase of the pressure caused by the algometer, even when it failed to produce pain.

(*e*) *Acuæsthesia.*—The head of a pin could be correctly discriminated from the point everywhere over the analgesic area.

(*f*) *Appreciation of difference in size.*—Tested by cylindrical metal rods with diameters of 2 cm., 0·75 cm. and 0·2 cm., he was equally accurate in his answers on both halves of the body.

(*g*) *The vibrations of a tuning-fork* (128) were accurately appreciated everywhere. There was no difference between the two halves of the body.

(*h*) *The sense of passive position* was perfect in both great toes, both ankles, both knees, and at all the joints of both upper limbs.

(*i*) *The compass-test.*—This was difficult to apply, owing to the violent reflex movements which were produced when the two points were brought into contact with the skin over any part of the analgesic and thermo-anæsthetic area, particularly over the sole of the left foot. This made it impossible to determine the threshold with any accuracy on the left side. But, when the points were applied carefully to the left sole and those answers only recorded that were unaccompanied by a violent reflex movement, we could not assure ourselves that there was any difference between the response from the right or the left foot.

(*j*) *Tactile localisation* was unusually accurate, and we could find no difference between the two sides of the body.

Cranial nerves.—The pupils reacted well to light and to shade. The movements of the eyes, face and tongue were perfectly performed.

Spine.—A radiograph of the spine showed disorganisation of the sixth and seventh cervical vertebræ.

CASE 6.—JAMES M.

He injured his spine by diving into shallow water in June, 1886. He did not lose consciousness at once, but was delirious for six weeks.

He gradually recovered power, and in three years reached his present condition. He came to the London Hospital in December, 1906, for lead colic, and was found to be a characteristic instance of Brown-Séquard paralysis.

Right.	*Left.*
No motor disturbance.	*Wasting of small muscles of hand.*
Reflexes normal.	*Slight spasticity of leg.*
Loss of sensation to pain, heat, and cold (Figs. 45 & 46).	*Knee-jerk exaggerated.*
Tactile sensibility perfect.	*Ankle-clonus.*
	Plantar reflex, extensor.
	No change in sensation.
	Passive position and movement perfectly appreciated. Compass-test gave perfect results.

James M., aged 41, a painter, came to the London Hospital in December, 1906, complaining of an attack of lead colic. He was discovered to have the symptoms of Brown-Séquard paralysis, of which he gave the following history.

In June, 1886, he was in the Army and was stationed at Aden. It was the custom of those men who could swim to bathe in the evening from Steamer Point. So large were the waves that it was possible to dive into them with safety. But one evening in June when attempting this feat, he miscalculated his distance and, diving into shallow water, struck the sand with his head. All power went from his limbs immediately, but he did not lose consciousness even for a moment. He was swept back by the wave, but was brought to land and stretched out upon the sand. He

suffered no pain, but seemed as if swollen to four times his usual size. They carried him to hospital and put him to bed; then he went quietly to sleep and did not regain consciousness for six weeks. He was told that he was delirious all the time. When he came to himself he found that he had several bed-sores; he could move his hands a little but no other limb.

Slowly, power returned to the right leg and both arms. It was six months before he could stand and then the left leg dragged.

He was sent to Netley and remained there until June 26, 1887, able to walk but dragging the left leg.

After his discharge from the Army he found work, and in three years had reached exactly the condition in which he first came under our notice.

He is a foreman painter and is in charge of the mixing of the paint. Once or twice a year he has suffered from pains in the stomach, evidently slight attacks of lead colic; he comes to the hospital and rapidly recovers. It was on one of these visits that his condition was discovered by Mr. Sherren, who transferred him to us.

Condition in December, 1906.

He is an intelligent man, temperate in his habits and answers well to sensory tests. He thinks there is nothing amiss with him, but was content to lie still in bed under observation as often as he was wanted, provided arrangements were made for his men to continue their work.

Motion.—He could stand with his eyes open and shut, and was in no way ataxic.

When he walked, the left leg was held a little stiffly, and he said he could not run, "because the left leg would catch." So long as he walked slowly, the toe cleared the ground completely. All the movements of both lower extremities were strong against resistance and there was no paralysis of any group of muscles.

He said that the left leg occasionally "jumped" at night; this never happened to the right leg.

The small muscles of the left hand were definitely wasted; all the interosseous spaces were hollowed and the hypothenar eminence was unnaturally small. The outer group of thenar muscles, formed by the opponens and abductor, was well developed, but the remaining muscles of the thumb were wasted compared with those of the opposite hand. He could strongly abduct and oppose the thumb, and adduction and flexion were possible; extension was perfect.

The grasp of the left hand was 2 kilograms, compared with a dynamometer reading of 25 kilograms from the right hand. This profound difference in the two hands was produced by the difficulty with which he grasped the instrument, in consequence of the want of power to flex the metacarpo-phalangeal joints strongly; and yet, when he made this movement apart from the dynamometer, it was carried out perfectly. The loss of grasp, when he attempted to exert pressure by flexing the fingers into the palm, seemed to be due to weakness of the interossei. The fingers could not be held out straight without falling out of alignment, and the interossei and lumbricales were acting feebly, if at all. All the long

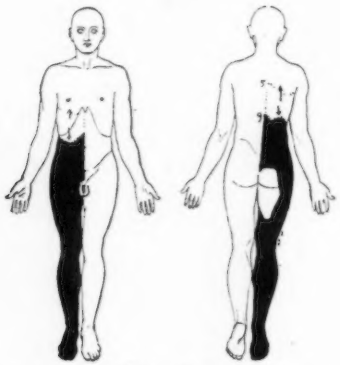


FIG. 45.

To show the loss of sensation to prick in Case 6. The line marked with an arrow pointing upwards corresponds to the border of the cutaneous analgesia discovered by testing from insensitive to sensitive parts. The border marked with an arrow pointing downwards shows the limits of the sensibility to pain when he was tested from normal to insensitive parts.

flexors and extensors acted well, and there was no loss of power in the muscles of the elbow and shoulder.

The muscles of the right upper extremity reacted well to both currents. The only changes in electrical irritability were found in the small muscles of the left hand; the interossei and the abductor minimus digiti did not respond to the interrupted or constant currents. The outer thenar muscles reacted briskly and normally, but the inner group contracted poorly to both forms of electrical stimulation.

Reflexes.—Wrist- and elbow-jerks were equal on the two sides. But the left knee-jerk was much exaggerated. Ankle-clonus was

obtained and the plantar reflex on this side gave an extensor response.

On the right side, the knee-jerk and plantar reflex were normal and no ankle-clonus could be obtained.

Sensation.—Except for the slight colic, which rapidly passed away, he complained of no abnormal sensations of any kind. But he found that, when the weather grew cold, he lost the use of his left hand, although in warm weather he could notice little difference between the two hands.

(*a*) *Sensibility to pain.*—To all cutaneous painful stimuli, such as the prick of a pin, sensation was lost over the area on the right half of the body shown in Fig. 45. The frontal boundary of this analgesia varied according to the method of testing. If the stimulus travelled from parts that were insensitive to those of normal sensibility, the analgesia extended up to the higher of the two lines; but, when the testing began over normal parts, he still responded to the painful stimulus, until the second of the two lines was passed. This is a characteristic feature of areas which are partly insensitive only.

Another remarkable feature of this case was the maintenance of sensation to painful cutaneous stimuli over a patch upon the right buttock and over the whole of the penis and scrotum.

The pain of excessive pressure was greatly diminished over the right sole and right leg below the knee. The algometer gave the following readings.

	R.	L.
Sole	Pain at 14	Pain at 4.
Inner aspect of leg	Pain at 8	Pain at 2·5.
Outer aspect of leg	Pain at 15	Pain at 7.
Calf	No pain at 12	Pain at 8.
Knee	No pain at 13	Pain at 5.
Front of thigh	Pain at 7	Pain at 6.
Ischial tuberosity	Pain at 8	Pain at 5.
Iliac crest	Pain at 4	Pain at 4.
Sterno-clavicular joint	Pain at 2	Pain at 2.

When a strong current was applied to the right or analgesic leg, he complained that it was "uncomfortable but not painful." Then the same current was applied quickly to the left calf and immediately withdrawn on account of the pain; but this stimulation had caused "cramp" in the left calf and he sat up in bed, rubbing his leg. Thus it is evident that the deep muscular pain upon which "cramp" depends must have been abolished in the right leg. This agrees with the results obtained with the algometer.

(*b*) *Thermal sensibility.*—He was insensitive to all degrees of cold over the area shown on Fig. 46. The extent of anæsthesia to all degrees of heat almost exactly corresponded with that for cold.

(*c*) *Tactile sensibility* was perfectly preserved; cotton wool was everywhere recognised, and No. 5 (360 mgm.) of von Frey's hairs produced a sensation of equal intensity over the great toes and over the sole and dorsum of both feet.

(*d*) *The vibrations of a tuning-fork* (128) were perfectly appreciated everywhere.

(*e*) *Acuæsthesia.* — He could tell the difference between the head and the point of a pin everywhere. In fact he is so clever

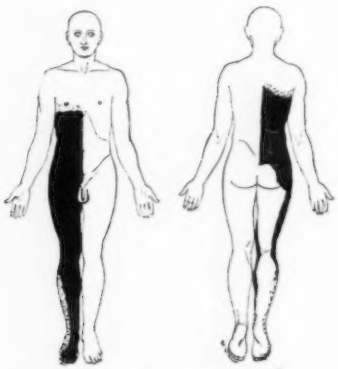


FIG. 46.

To show the extent of the loss of sensibility to thermal stimuli in Case 6.

at telling the head from the point that it is sometimes difficult to work out the loss of painful cutaneous sensibility by means of a pin.

(*f*) *Sense of passive position and movement* was everywhere perfect. His answers in response to movements of either foot were unusually quick and certain. So trustworthy was he that throughout the testing of the great toes, ankles, knees, elbows, wrists, thumb, index and middle fingers, he did not make a single mistake, although 20 movements were made at each joint (320 stimulations).

(*g*) *The Compass-test.*—He gave perfect answers over the palms at 2 cm. and over the forearms at 4 cm. distance. When

the compasses were applied longitudinally to the sole of the foot, his answers were perfect at 4 cm., but were almost equally faulty at 3 cm.

Right 3 cm. $\frac{1 \mid 9\text{ R. }1\text{ W.}}{2 \mid 8\text{ R. }2\text{ W.}}$ Left 3 cm. $\frac{1 \mid 7\text{ R. }3\text{ W.}}{2 \mid 9\text{ R. }1\text{ W.}}$

Thus, there was certainly no difference in the sensibility of the two soles to this test.

(*h*) *Tactile localisation* was equally good on the two halves of the body. When asked to name the spot touched, he did so correctly in every case, even when the touch was produced with No. 8 (830 mgm.) of von Frey's hairs.

Tested by Spearman's method (*vide* pp. 548 and 627), the results were as follows: Six touches were given at each spot and the numbers represent the average deviation of his six attempts to point to the spot stimulated:—

	R.	L.
Leg	2·6	2·9.
Dorsum of foot	4·2	6·2.

The difference between the two sides was, we believe, within the experimental error.

Cranial nerves.—The pupils reacted well, and there was no abnormality in the function of any cranial nerve.

Spine.—A radiograph showed no abnormality of any kind.

Sphincters.—He could hold his water for half an hour, but it occasionally dribbled away a little if he attempted to hold it too long. So long as he remained constipated, he could hold his motions; but when they became loose, he lost all control.

Sexual.—He has no desire during the winter months; sometimes during the summer he feels desire, but has never had an erection or emission since his accident. After leaving the Army, he married "for the sake of a home," and has no children.

Case 7.—Cyril Wilfred F.

August 14, 1905, *he dived into shallow water and injured his spine. Sensation was affected in the left leg, and, after the effects of shock had passed away, his right arm remained weak.*

Seen in November, 1905, *there was no definite motor paresis, but the reflexes were brisker on the right side than on the left. Sensibility to pain, to cold and to heat were lost to a different extent over the left leg and left half of the body* (*Figs.* 47, 48, & 49).

No other sensory function was disturbed. A radiograph showed changes in the body of the sixth cervical vertebra.

In 1906, *observations were made on the behaviour of the area on the trunk insensitive to heat, but sensitive to cold, in order to elucidate the condition known as paradox cold.*

Cyril Wilfred F., aged 26, of no occupation.

On August 14, 1905, whilst diving from the top of a bathing-machine, he slipped, pitching head first into shallow water. He believes that the top of his head struck the sand. He floated to the top, and did not lose consciousness, but found he was unable to move his arms or his legs. Power returned in all his limbs within half an hour, but he remained in bed for three weeks,

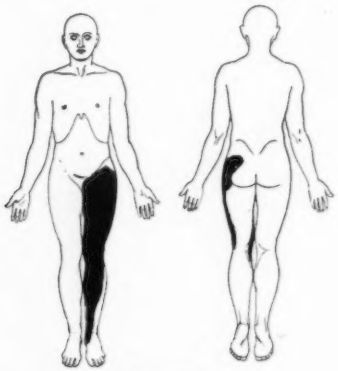


FIG. 47.

To show the loss of sensibility to cutaneous painful stimuli in Case 7.

suffering from a good deal of pain in the neck. For nearly three months he could not use the right arm, and, from the beginning, noticed that sensation was affected in the left leg, particularly to heat and cold. At first the neck was stiff, and he suffered from pain, especially on movement, at a level of about the seventh cervical vertebra.

Condition in November, 1905.

He came under our notice in November, 1905, exactly three months after the accident. His condition then was as follows.

He was a small, well-built, muscular man, and showed no signs of any disease, except in the nervous system.

Motion.—He walked well, and could stand perfectly with his eyes closed. There was no sign of paralysis, or spasticity in the legs. The grasp of the left hand was distinctly more powerful than that of the right, but there was no wasting or paralysis in either upper limb. He could touch his nose perfectly with either hand, even when his eyes were closed.

Reflexes.—The right wrist-jerk was slightly brisker than the left, and the knee-jerk was distinctly more active on the right side than on the left. No ankle-clonus was present on either side. When testing for the plantar reflex, the left great toe gave a decided flexor response, but the right remained stationary.

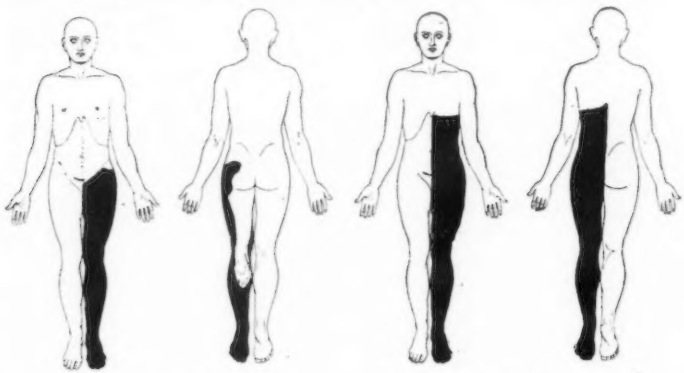


FIG. 48. FIG. 49.

To show the loss of sensation to cold (Fig. 48) and to heat (Fig. 49) in Case 7. The area on Fig. 48 enclosed by a dotted line was sensitive to cold, but not to heat. It was here that the observations were made on the production of paradox cold by temperatures of 45° C. and above.

Sensation. (*a*) *Sensibility to painful stimuli.*—Sensation was profoundly affected over the left lower extremity. Over the area shown in Fig. 47, he was entirely insensitive to the painful aspect of a pin prick; but over the sole of the foot and over the back of the leg he was certainly sensitive to painful cutaneous stimuli. In the direction of the trunk, and also towards the dorsum of the foot, the boundaries of this area of complete cutaneous analgesia shaded gradually into parts of normal sensibility.

(*b*) *Thermal sensibility.*—Over the area in Fig. 48, he was

insensitive to a test-tube containing ice water, but the borders of this area were not rigidly defined, excepting over the outer side of the thigh. Within these parts, he was also insensitive to all temperatures which caused a sensation of coolness over the normal leg.

Temperatures of from 30° to 70° C. produced no sensation of heat over the area in Fig. 49. But he complained that with a stimulus of above 50° C. pain was produced over all those parts insensitive to heat, but sensitive to cutaneous painful stimuli. Thus, it was possible to map out the area of cutaneous analgesia with a test-tube containing water at 60° C.

(*c*) *Tactile sensibility* was everywhere perfect, both to light and deep touch.

(*d*) The *vibrations of a tuning-fork* were everywhere accurately appreciated.

(*e*) The *sense of passive position and movement* was nowhere disturbed.

(*f*) To the *compass-test* his answers were equally good over both halves of his body.

(*g*) *Cutaneous localisation* was equally good over synonymous parts of the body and limbs.

Spine.—A radiograph showed changes in the body of the sixth cervical vertebra.

Condition in October, 1906.

Except that his general health improved, his condition remained unchanged. But, as some additional observations of interest were made in the autumn of 1906, especially those relating to paradox cold, we shall recapitulate shortly his condition at that time.

Motion.—He walked with such ease that he asserted he was as well as ever. The grasps of the two hands were of equal strength.

Reflexes.—The right wrist-jerk was perhaps slightly more active than the left, and the knee-jerk was certainly brisker on the right side than the left. No ankle-clonus could be obtained, and whilst the left plantar reflex was certainly flexor in type, the right was indeterminable.

Sensation. (*a*) *Sensibility to painful stimuli.*—The loss of sensation to the prick of a pin and to the painful interrupted current corresponded exactly to that on Fig. 47.

The algometer gave the following readings, showing how closely the decrease in sensibility to the painful aspect of excessive pressure corresponded with the cutaneous analgesia (compare Fig. 47 with the algometer readings).

	R. (normal).	L. (sensory loss).
Sole	Pain at 2·5.	Pain at 2·5.
Shin (inner aspect)	Pain at 1·5.	Pain at 10.
Calf	Pain at 4.	Pain at 5.
Front of knee	Pain at 1·5.	Pain at 10.
Popliteal space	Pain at 2·5.	Pain at 5.
Front of thigh	Pain at 4·5.	Pain at 11.
Ischial tuberosity	Pain at 3.	Pain at 4·5.
External to the anterior superior spine of the ileum :—		
(*a*) Above the line of cutaneous analgesia	Pain at 2·5.	Pain at 2·5.
(*b*) Below the line of cutaneous analgesia	Pain at 2.	Pain at 5.
Palm of the hand	Pain at 2·5.	Pain at 2·5.

(*b*) *Thermal sensibility.* — Some striking experiments were made to elucidate the phenomenon known as "paradox cold." The area insensitive to all cold stimuli was of considerably less extent than that insensitive to heat (Fig. 48 and Fig. 49). Thus, the whole of the abdomen and lower part of the thorax on the left side enclosed by a dotted line on Fig. 48 was sensitive to cold, but not to heat. So long as the stimulus was 40° C. or below, it produced no sensation over this area insensitive to heat; it was called in every instance "a touch." But, as soon as temperatures of over 45° C. were employed, the patient invariably said the stimulus was "cold." If the temperature was raised to 55° C., he complained of "cold pain."

These observations show that, in this instance, the paradox cold obtained by the application of heat to an area insensitive to heat was due to stimulation of cold spots. Such spots reach normally to temperatures above about 40° with the production of a sensation of coldness.

(*c to g*).—Every other sensory quality was perfect, as was the case in 1905.

CASE 8.—G. G. A.

In August, 1904, *whilst in India, he began to suffer from fever: this continued till November.*

January 31, 1905, *he was suddenly seized with violent pain in the neck, and lost power in the left arm and left leg, together with sensibility to pain, heat and cold over the right leg and right half of the body.*

When first seen in April, 1905, *he was a characteristic instance of Brown-Séquard paralysis.*

Right.	*Left.*
No motor disturbance.	*Slight spasticity of left leg.*
	Slight wasting of left hand.
Knee-jerk normal.	*Knee-jerk greatly exaggerated.*
No ankle-clonus.	*Ankle-clonus.*
Plantar reflex flexor in type.	*Plantar reflex extensor in type.*
Loss of sensibility to cutaneous painful stimuli (*Fig.* 50) *and to cold* (*Fig.* 51).	*Sensation perfect.*
No loss to heat.	
All other forms of sensation perfect.	

G. G. A., aged 45, a medical man, was in perfect health until August, 1904. He then began to suffer from pyrexia; his temperature never rose above 100° F. (37° C.), but never remained normal for long. He was in India at the time, and the condition was supposed to be malarial, but quinine was entirely without effect. The pyrexia continued until the middle of November, when he thought he was well and resumed his usual duties.

On January 31, 1905, he was suddenly seized with violent pain slightly to the left of the spine of the seventh cervical vertebra: it radiated down the back of the shoulder and up the neck and was worse at night than in the day. This pain increased rapidly for two weeks and his temperature rose again, rising and falling each day as before.

Then the pain became easier, and he was able to sit up and play the banjo. Within two days, his left arm became paralysed, but the pain continued to decrease in severity. The paralysis spread to the left leg, and at the same time the whole right half of the body as high as the nipple became insensitive to pain, heat and cold.

At no time was the motor power of his right leg diminished, and he had no difficulty in passing or in holding his water.

The right half of the face did not sweat.

About the middle of March, 1905, the paralysis began to pass away from the left arm. By the middle of April, 1905, his temperature ceased to rise above 99° F. (37·2° C.), and since that date he has steadily improved.

At the age of 18 he had an attack of venereal disease under a long foreskin; this produced phimosis, for which he was circum-

cised. Otherwise he was not treated and suffered no consequences.

Condition in April, 1905.

When he was first seen by one of us in April, 1905, he was a well-built active man, in appearance younger than his age.

There were no abnormal signs in the heart, lungs or abdomen; the spleen was not enlarged and his urine was normal.

Motion. — He walked a little stiffly, in consequence of slight spasticity of the left leg, but could stand firmly on either leg and did not fall when his eyes were closed.

The grasp of the left hand was greater than that of the right. All the interossei of the left hand were a little wasted, especially

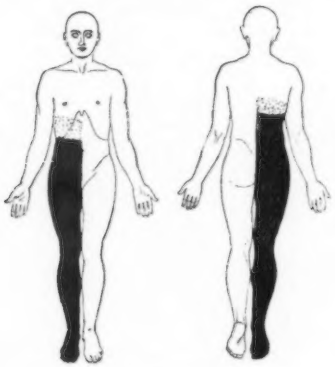


FIG. 50.

To show the loss of sensibility to cutaneous painful stimuli in Case 8. The dotted area represents a band of diminished sensibility.

the first, and the fingers were a little over-extended at the metacarpo-phalangeal, and flexed at the interphalangeal joints. The thenar muscles showed slight signs only of wasting. All the muscles reacted perfectly to the interrupted and constant currents.

Reflexes.—The left knee-jerk was greatly increased compared with the right, and ankle-clonus was definitely present on the left side; no clonus could be obtained from the right ankle. On the left side, the plantar reflex was of the extensor type, whilst that from the right foot gave a flexor response.

Sensation. — He complained of no abnormal sensation, unless the right leg was touched with a cold object; for instance, when

standing on a cold floor, he noticed a luke-warm sensation in the right foot instead of the normal coldness.

(*a*) *Sensibility to pain.*— He was completely insensitive to all cutaneous painful stimuli over the area in Fig. 50. The frontal border was ill-defined, merging into parts of normal sensibility, and the boundary of the analgesia differed according to whether testing was carried out from normal to abnormal parts or *vice versâ*.

The right testicle and right half of the penis and scrotum were insensitive to painful stimuli.

(*b*) *Thermal sensibility.*—He showed a remarkable dissociation between sensibility to heat and to cold. All temperatures between 35° C. and 55° C. could be appreciated everywhere over

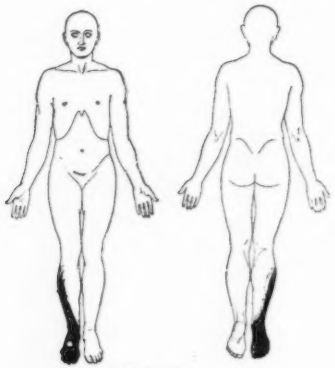


FIG. 51.

To show the loss of sensibility to cold in Case 8.

the right leg, and 35° C., 45° C., and 50° C. could be discriminated.

But, over the area marked on Fig. 51 he was entirely insensitive to all temperatures which normally produced a sensation of coldness.

(*c*) *Tactile sensibility* in all its forms was perfect, whether the stimulus was cotton wool or a deep touch.

(*d*) *Acuæsthesia.*—He could tell the head from the point of a pin everywhere over the body and limbs.

(*e*) The *sense of passive position and movement* was everywhere perfectly preserved.

(*f*) *Tactile localisation* was equally good over both lower extremities.

Spine.—He had great difficulty in turning his head to the left until a few weeks before he was first seen. Even then he could not turn it rapidly. The radiograph gave results that were uncertain, all that could be said was, that the shadow was somewhat more blurred on the left side than on the right in the region of the transverse processes of the fifth and sixth cervical vertebræ.

Sphincters.—He has had no trouble with micturition and defæcation.

Condition in October, 1906.

He greatly improved, and was last seen in October, 1906.

Motion.—The left leg no longer gave him trouble, and was not stiff or spastic; he could run upstairs "two at a time."

Reflexes.—The reflexes were unchanged, still greatly increased in the left lower extremity.

Sensation. (*a*) *Sensibility to pain.*—The area insensitive to prick had greatly decreased, and, although over the back and front of the right thigh and over the sole of the foot the sensation was diminished, the only absolute loss of cutaneous painful sensibility lay over the leg between the knee and the ankle.

This was associated with a diminution in sensibility to the pain of pressure compared with the left leg, as shown by the following algometer readings:—

	R. (sensory loss).	L. (motor).
Sole	Pain at 7·6·5	Pain at 2·2·5.
Shin (inner aspect)	Pain at 4	Pain at 1·5·2.
Calf	Pain at 5·6	Pain at 3·3·5.
Knee	Pain at 10·7	Pain at 4·3.
Popliteal space	Pain at 3·4	Pain at 2·5·2·5.
Iliac crest	Pain at 7·6	Pain at 3·3.
Buttocks	Pain at 3·3	Pain at 2·5·2.
Lower ribs	Pain at 1·5	Pain at 1·5.
Palm	Pain at 3·3	Pain at 3·3.

These readings show no absolute loss of sensibility to the pain of pressure, but all those from the right leg are uniformly somewhat higher than those from similar parts of the left leg. Two readings are given to show the manner in which the algometer results vary when sensation is not abolished.

(*b*) *Thermal sensibility.*—Anæsthesia to cold was still present over the external aspect of the right leg and dorsum of the foot, but no longer occupied the sole. Over this area of loss of sensation, water at 20° C. and below was uniformly called "warm."

(*c-f*).—Every other sensory function was perfect.

Case 9.—Joseph F.

A glio-sarcoma of the central gray matter of the spinal cord extended from the medulla to the seventh thoracic segment. Situated for the greater part in the centre of the cord, it had invaded and destroyed the left half in the region from the third to the seventh cervical segments.

It was found on his admission to the hospital that motion was lost in the left arm, but it was not until shortly before his death that the right arm became paralysed.

Both knee-jerks were increased; ankle-clonus was obtained on both sides, and both plantar reflexes gave an extensor response.

For some time after admission, the local disturbance of sensation consisted of analgesia and thermo-anæsthesia of the left upper extremity, with abolition of the sense of passive position and movement and of tactile discrimination. Tactile sensibility was preserved. Shortly before death, tactile sensibility was also lost in this arm.

The remote disturbance of sensation consisted throughout of loss of sensibility to pain, heat and cold over the right leg, right half of the body and right arm and hand.

The sense of passive position was greatly diminished in the left foot, but perfect on the right side.

Joseph F., aged 44, an actor and vocalist, was admitted to the London Hospital under the care of Dr. Dawson on July 6, 1905, and died on August 10.

In November, 1904, he first noticed slight weakness and stiffness in the left arm with a tingling sensation in the fingers of the left hand. Shortly afterwards his neck began to grow stiff.

Until February, 1905, he was able to play the piano with both hands, but the left arm grew steadily weaker; slight weakness appeared in the right arm just before admission.

On two occasions within the fortnight before admission he passed urine involuntarily.

He had suffered from no previous illness, except an attack of venereal disease, accompanied by warts, but followed by no secondary consequences. He had never taken alcohol to excess.

Condition shortly after Admission.

He was an intelligent man, interested, until shortly before his death, in his condition and the testing of his sensation. His

speech was unaffected, and he never suffered from fits or headache.

Motion.—The left arm lay flaccid and motionless on the bed beside him. The small muscles of the left hand were wasted, and the left arm was nearly 1·5 cm. smaller in diameter than the right. No fibrillary twitchings were visible, and the muscles of the left arm and hand reacted perfectly both to the interrupted and to the constant current.

The small muscles of the right hand were wasted and the grasp was weak; but all movements of the thumb and fingers and of the rest of the upper extremity could be made without difficulty.

Both legs were distinctly spastic but were not paralysed; the muscles were flabby but not wasted.

Reflexes.—Both knee-jerks were increased, ankle-clonus was obtained on both sides, and both plantar reflexes gave an extensor response.

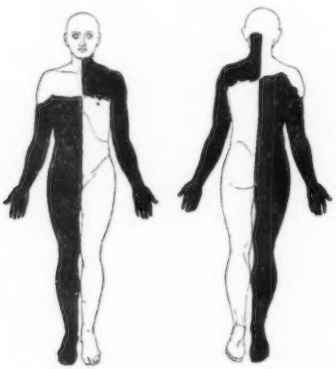


FIG 52.
To show the loss of sensation to cutaneous painful stimuli in Case 9.

Sensation.—(*a*) *Sensibility to pain.*—He was insensitive to all cutaneous painful stimuli, such as the prick of a pin or the interrupted current, over the whole of the right leg, right half of the body and right arm (Fig. 52). On the opposite side, the analgesia occupied the left arm and left half of the neck.

Excessive pressure produced no pain over either palm, but the amount of the pressure applied was not measured with the algometer.

(*b*) *Thermal sensibility.*—Sensibility to heat and cold was

abolished over the area shown in black on Fig. 53. Over the right side of the neck it was greatly diminished, more so to heat than to cold.

(*c*) *Tactile sensibility* was unimpaired at this time; even cotton wool was appreciated over the left arm and hand.

(*d*) *Sense of passive position and movement.*—He was unable to recognise the position of any part of the left upper extremity, and was ignorant of even the grossest movements; but in the right hand and arm the sense of passive movement and position was perfect.

His power of recognising movements of the left great toe was defective, but from the ankle and knee his answers were correct.

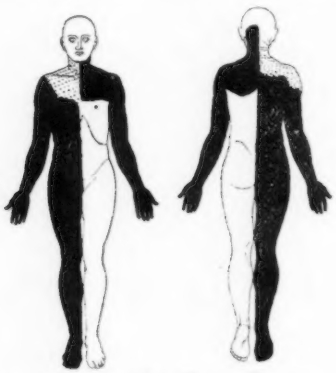


FIG. 53.
To show the loss of sensation to heat and cold in Case 9.

On the right side he was uniformly accurate in his appreciation of movements of the great toe, ankle and knee.

(*e*) *Compass-test.*—Tactile discrimination was perfect in the right hand, and he made two mistakes only when the compass-points were separated to a distance of 1 cm. But over the left hand the two points were called one even when 5 cm. apart.

		L.		R.
Palm (applied transversely)	5 cm.	1 | 10 R. 2 | 10 W.	2 cm.	1 | 10 R 2 | 10 R.
			1 cm.	1 | 9 R. 1 W. 2 | 9 R. 1 W.

Cranial nerves.—The pupils reacted to light and accommodation, and the ocular movements were good. There was no paralysis of the face, and the tongue was protruded well. He could move both halves of the palate and at this time swallowed well.

Subsequent progress.—He gradually lost power in the right arm and hand, and although every movement could be carried out feebly, could at last no longer feed himself. Tactile sensibility became so greatly diminished in the left arm that the compass-test could not be applied; he gave no answer when touched with the two points. Over the whole of the left upper extremity, he now gave no response to the pressure with a pin, whereas previously every prick was said to be a touch.

The extent of the insensibility to pain, heat and cold remained as before.

Gradually complete incontinence of urine and fæces came on and swallowing became difficult.

By August 9 the right arm was completely paralysed and on the 10th he died suddenly.

Post-mortem Examination.

An autopsy was made on August 11. Rigor mortis was present. No abnormalities were found in the lungs, heart, liver, kidneys, or spleen.

The brain and spinal cord were removed together by sawing through the occipital bone. The spine was found to be entirely unaffected. But it was at once obvious that the spinal cord was greatly enlarged in the cervical region, and this increase in size extended up to the bulb. During the manipulations a considerable quantity of clear fluid escaped.

No changes were found in the cerebrum or cerebellum.

On microscopical examination a growth was found extending from the region of the olive to the eighth thoracic segment, situated in the gray matter around the central canal. But, even at levels as low as the third lumbar, the tissue around the central canal was abnormally extensive. Throughout the cord, the growth lay in the gray matter forming at the first and second cervical level a well-defined round tumour in the centre of the spinal cord. But, at the third cervical level, it had invaded and destroyed the whole of the left half of the spinal cord; the right half was much compressed. This destruction, more particularly of the left half of the cord, extended to the level of the seventh cervical. Below this, the tumour again assumed a more central position and could be clearly seen to the seventh thoracic level. Below this, some increase in the tissue around the central canal alone marked the position occupied by the tumour in higher segments. When stained by the Weigert-Pal method the whole of the crescentric portion representing the displaced right half of

the spinal cord on Figs. 1 and 2 [Plate 1] took the stain well, and showed large numbers of apparently normal fibres. In the dorsal region this stain showed profound degeneration of the pyramidal trace in the left half of the cord; in the right half this tract took the blue stain well. [Plate 1.]

The growth consisted of cells with a large deeply-staining nucleus and a small amount of protoplasm. Heidenhain's iron-hæmatoxylin method showed that many of these cells possessed processes which interlaced. It was probably a glio-sarcoma starting in the tissue around the central canal. Hæmorrhage had taken place at several points within the growth.

CASE 10.—JAMES Y.

July, 1906, *he began to lose power in his legs, first in the left and then in his right; at the same time he ceased to be able to pass his water, and developed retention of urine with overflow.*

Admitted to Hospital with a high temperature and signs of bronchitis in both lungs.

He improved and between August 10 *and* 15 *complete examination was possible.*

Absolute paralysis of motion was present below the umbilicus.

There was much loss of sensation to painful and thermal stimuli over both lower extremities (*Fig.* 54).

Sensibility to light touch was perfect except over left foot.

He had lost all appreciation of passive position and movement in the lower limbs, and was unable to discriminate two points separated to more than twice the normal distance.

Yet he could name the spot touched accurately, although ignorant of the position of his legs.

On post-mortem examination, a metastatic growth secondary to a hypernephroma of the right kidney was found in the left half of the spinal cord. This had caused hæmorrhage, which extended widely in the left half of the cord from the seventh thoracic to the second lumbar segment.

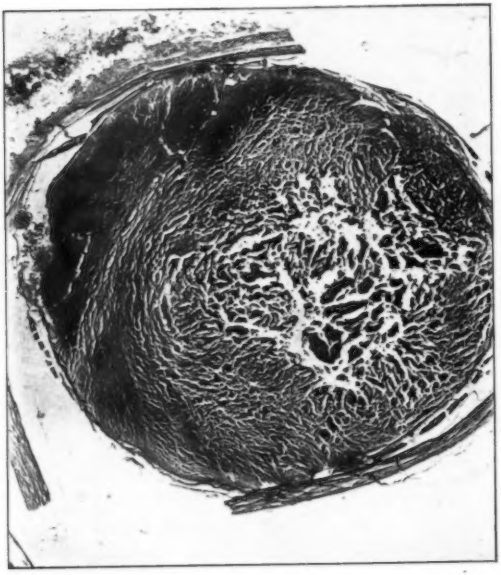
Profound acute degeneration was found in both posterior columns, in both lateral bundles of Gowers and in both direct cerebellar tracts.

James Y., aged 61, was admitted to the London Hospital on July 27, 1906 (Dr. Schorstein).

For about a month he had complained of pains in the back, chiefly in the lower dorsal and upper lumbar region. Three

PLATE I.

Right.



Left.

FIG. 1.—Cervical 3.

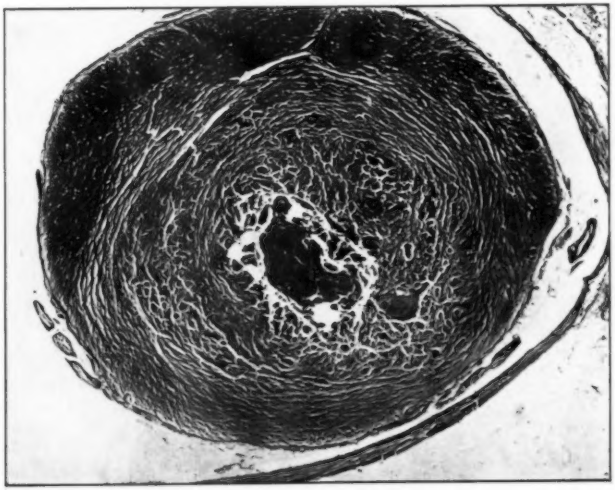


FIG. 2.—Cervical 6.

weeks before admission he began to lose power, first in the left and then in the right leg. At the same time he noticed that he was unable to pass water, and yet as he stood it would run from him. Occasionally, during the three weeks before admission, he had lost control over his motions.

He was a file-cutter, but had never suffered from any form of lead poisoning. No history of venereal disease could be obtained; he had been married for 34 years and had five healthy children. He denied all excess of alcohol, and had the appearance of a man of temperate habits.

Condition on Admission.

When first admitted in July, 1906, his temperature ranged between 100·2° F. and 102·4° F. (37·8° C. and 39° C.). He lay propped up in bed with flushed cheeks, breathing rapidly; the pulse was 120 in the minute and regular. Both lungs were emphysematous and râles were widely heard, but at first no signs of consolidation were discovered. The heart-sounds were in no way abnormal. The bladder was distended almost to the umbilicus, and the urine drawn off by catheter was faintly acid and contained some mucus and urates. Over the sacrum was a large bed-sore.

Progress.—During the first fortnight his urine was drawn off regularly, and he rapidly improved under treatment. The temperature gradually fell, so that from August 10 to August 15 it did not rise above 99° F. (37·2° C.). During this period he was so much better that careful observations could be made upon the condition of sensation; he became cheerful, interested in the testing of sensation, and remarkably trustworthy in his answers, if precautions were taken that he should not become exhausted.

But on August 15, the temperature began to rise and on the 16th he had a rigor. Further rigors followed on the 18th; definite signs of pneumonia appeared in the right lung, and he died on August 20.

Observations on the Condition of the Nervous System made between August 10 *and August* 15.

The patient was intelligent and answered well to the control-tests applied. At the time the following observations were made, his temperature was normal. He was bright and cheerful, with the hope that he was on the way to recovery.

Sensation.—He complained of no pain or abnormal sensations in the lower extremities, which were alone the seat of gross sensory defects.

(*a*) *Pain.*—The prick of a pin was not appreciated over the area shown in Fig. 54. Below the knees, the loss of sensation was absolute, merging gradually above into parts of normal sensibility. Over the front of both thighs he could appreciate the pain produced by a prick, but sensation appeared to be diminished; above the level of the groin it was entirely unaffected.

This distribution exactly agreed with the analgesia to the painful interrupted current. Over both lower extremities below the level of the groin, sensation was somewhat diminished, but the only complete loss of sensation to this stimulus corresponded to the parts marked on Fig. 54.

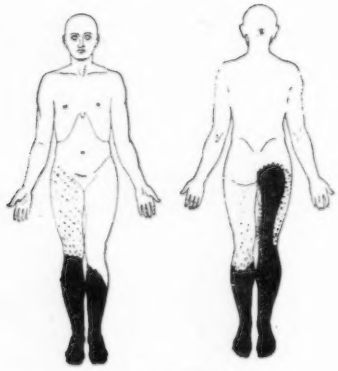


FIG. 54.

To show the loss of sensation to cutaneous painful stimuli in Case 10.

The algometer showed that pain was produced when pressure reached the following amount, measured in kilograms.

	R.	L.
Sole of foot	10 (dull pain)	10 (dull pain).
External malleolus	7	7.
Inner aspect of tibia	5	5.
Inner aspect of knee	6	6.
External to the anterior superior spine of the ileum	6	6.
Head of metacarpal bone of thumb	5 (sharp pain)	5 (sharp pain).

(*b*) *Heat and cold.*—The parts insensitive to a temperature of 50°-55° C. corresponded to the blackened portions of Fig. 54; but the whole of the right thigh was also insensitive to water at 40° C.

The loss of sensation to temperatures between 0° and 25° C. corresponded on the left lower extremity with the anæsthesia to heat. On the right leg, the loss of sensation extended up to the groin; over the right thigh, stimulation with ice-cold water was invariably said to be "hot." Thus a series of tests with silver tubes, containing water at different temperatures, gave the following results: ice was called "hot," a temperature neutral to the normal skin was called "a touch," 55° C. appeared to be hot, 40° C. was said to be "a touch." At the time these tests were made, the temperature of the room was 24° C.

Here we find an instance of the rule that, when one of the two qualities of thermal sensibility is abolished, extreme degrees will be called by the name of the range of temperature which can be appreciated. Sensibility to cold (0° to 25° C.) was abolished over the right thigh, although 50° C. could be appreciated; the application of ice-cold water to this thigh invariably evoked the statement that the stimulus was hot.

(*c*) *Light touch.*—When first examined there seemed to be no part of either lower limb where he could not appreciate a light touch with cotton wool. But, on August 13th, he was somewhat insensitive to this stimulus over the left foot below the level of the ankle; elsewhere, however, even on this date he showed no trace of diminished sensibility to cotton-wool.

(*d*) *Deep touch and pressure* were everywhere appreciated, and he never failed to answer correctly throughout the whole period he was under examination.

(*e*) *Passive position and movement.*—He was entirely unable to recognise the position into which his lower limbs had been placed passively, and was unconscious that they had been moved. Extensive movements could be made at the ankles, knees and hips without his knowledge. If his eyes were closed, the legs could be moved from the extended position in any direction and the knee flexed through 40°, and he still imagined they lay stretched out before him on the bed. When he was allowed to open his eyes, his expression of surprise amply testified to the greatness of his error.

He was equally unable to recognise movements of the feet or legs produced by electrical stimulation of the muscles of the lower limb.

The appreciation of passive position and movement was not disturbed in any part of the body, except from the hips downwards.

(*f*) *Compass-test.*—Over the palm of the left hand, which we used as a control, he gave perfect answers when the compass

points were separated by 1·5 cm. and applied longitudinally. But, over the outer aspect of the left leg, he failed entirely at 15 cm., and over the front of the left thigh when they were 20 cm. apart.

Outer aspect of left leg. { 15 cm. $\frac{1 \mid}{2 \mid 1.}$ XX. 1D. DXD. 1XO. / XI. 11X. 11D. 1.

or $\frac{1 \mid 2 \text{ Right.}}{2 \mid 7 \text{ Right.}}$ 3 Doubtful. 4 Wrong, and once no answer. / 1 Doubtful. 2 Wrong.

Front of left thigh. { 20 cm. $\frac{1 \mid}{2 \mid 1.}$ XO. XXX. D. 11D. / 11. 1. XD. DXXD.

or $\frac{1 \mid 2 \text{ Right.}}{2 \mid 4 \text{ Right.}}$ 2 Doubtful. 4 Wrong, and once no answer. / 3 Doubtful. 3 Wrong.

(*g*) *Cutaneous localisation.*—On several occasions we satisfied ourselves that he was able to name the spot touched with accuracy; but one sitting was devoted to the following experiments.

(1) He was allowed to see the position of his limbs before they were touched. Then his eyes were closed and he was touched at similar spots on the two lower limbs, on the sole, the instep, the dorsum of the foot and just below the knee. In every case he named correctly the part touched.

(2) His eyes remained closed and his legs were moved so that he was unable to say in what position they lay. Then he was touched on the sole, the dorsum of the foot, the calf and just below the knee, first on the one limb and then on the other. In every instance his answers were correct. In fact, no difference could be discovered in the ease with which he named the spot touched when he knew how his legs were lying, and when he was totally ignorant of the position into which they had been placed.

He was never mistaken in naming the side to which the limb belonged that had been touched.

(3) His eyes were closed and the limbs were moved so that the left leg was flexed at the knee and hip, the right leg extended and abducted. Various points on the legs were touched and he was asked to indicate the spot with a light cardboard rod. The rod struck the bed idly; for so great was the error he was unable even to touch the legs, and yet he continued throughout to name correctly the spot touched.

(*h*) *Acuæsthesia.*—He was able to distinguish the head from the point of a pin all over both lower limbs, even over parts which were entirely insensitive to all painful stimuli.

(*i*) *Tuning-fork.*—The vibrations of a tuning-fork beating 128 were everywhere appreciated.

Motion.—Below the level of the umbilicus he was completely paralysed, and could make no movements of the hips, knees, ankles or toes. The legs lay flaccid, neither wasted nor spastic, all the muscles reacting normally to the interrupted and constant current.

Every other movement was perfectly performed and there was no wasting of the hands or arms.

Reflexes.—The knee-jerks were abolished, but ankle-clonus was obtained on both sides. Wrist and elbow-jerks were in every way normal.

The plantar reflexes gave an uncertain response.

Cranial nerves.—The pupils were equal and reacted to light and accommodation. Ocular movements were perfect. There was no affection of the face and tongue.

No changes were found on ophthalmoscopic examination.

Sphincters.—On admission to the hospital, his bladder was greatly distended and he never regained the power of passing his water. It was drawn off twice daily by catheter.

The bowels were obstinately confined, but when opened by enemata he was unable to retain his motions.

Post-mortem Examination (*August* 21).

The body had been preserved in the cool chamber at a temperature just above freezing point. Rigor mortis was present. Over the sacrum and buttocks was a large bed-sore which was present on the patient's admission to the hospital.

The right lung was almost solid from septic broncho-pneumonia, and within it, at the extreme base, was found a small abscess-cavity of about the size of a walnut. This lung was adherent to the chest-wall except at the base; here there was a small collection of pus.

The left lung was widely adherent and contained patches of septic broncho-pneumonia.

The larynx and trachea were healthy.

No abnormality was found in the pericardium or heart, but the aorta was somewhat atheromatous.

No fluid was found in the abdominal cavity. The spleen was much enlarged and weighed 14½ ozs. (411 grms.); its substance was softened. No abnormal changes were found in the liver, which was not enlarged.

On removing the right kidney a large encapsuled growth was seen adherent to and distorting its upper surface. This growth was about the size of a walnut, soft and of a light yellow colour.

On its inner aspect was seen the suprarenal capsule. In the lower part of the same kidney was a secondary growth, enclosed in a capsule, and of the same yellow colour.

On microscopical examination, the tumour was found to be a hypernephroma formed of columnar cells arranged in columns. Extensive hæmorrhage separated these columns of cells. Many of the cells contained brown granular material, apparently altered blood pigment.

The left kidney showed no gross abnormality.

No changes in the brain could be seen with the naked eye; it contained no secondary growths.

Condition of the spinal cord.—The cord was removed and hardened in 5 per cent. formalin. No growth or other abnormal changes were present in the membranes and no gross disease could be discovered before the cord was incised. But, as soon as it was cut across at the level of entry of each pair of roots, profound changes were at once visible, consisting firstly of hæmorrhage around a growth, and secondly of obvious degeneration in the posterior columns, which were whiter than the rest of the cord.

Distribution of the focal lesion (Plate 2).—The first sign of a focal lesion was found at the level of the seventh thoracic; here the base of the left posterior horn was occupied by an extravasation of blood. On both sides, the vessels of the posterior and lateral columns in the neighbourhood of this hæmorrhage were dilated and unduly visible.

At the level of the eighth thoracic, this hæmorrhage had spread so that it occupied the apex of the triangle formed by the left posterior column, but did not encroach on the lateral column.

At the ninth thoracic level, the hæmorrhage was rather more extensive, and for the first time dark granules could be seen amongst the extravasated blood.

At the level of the tenth thoracic, the lesion had reached such proportions that the whole of the left posterior horn and the greater part of the lateral column was destroyed. The external portions of the lateral column stained well by the Weigert-Pal method, but the whole of the blunt angle between the anterior and posterior horns was filled up with extravasated blood, which had also completely destroyed the central portion of the posterior horn. The posterior column was pressed upon and displaced, but took the Weigert stain well. Within the hæmorrhagic area were two foci, which consisted of small cells containing pigment. In sections stained by Van Gieson's method, the disease was seen to

PLATE II.

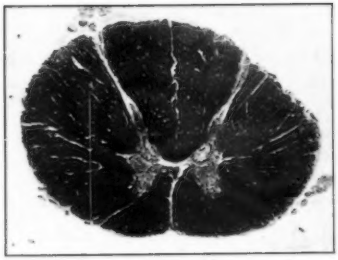


FIG. 1.—Thoracic 6.

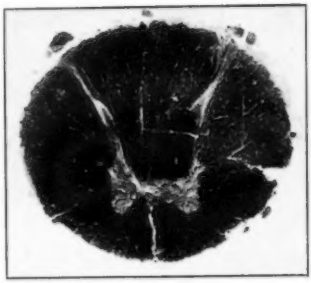


FIG. 2.—Thoracic 8.

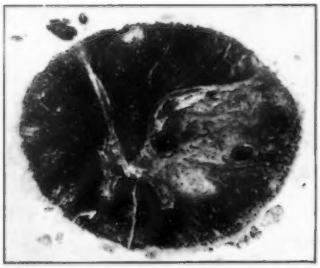


FIG. 3.—Thoracic 10.

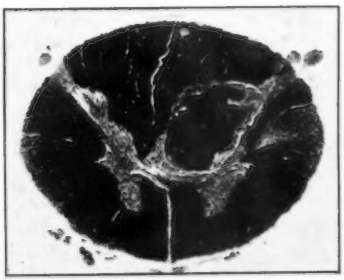


FIG. 4.—Dorsal 12.

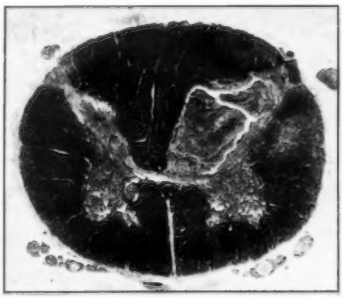


FIG. 5.—Lumbar 2.

PLATE III.

FIG. 1.—Cervical 4.

FIG. 2.—Thoracic 1.

FIG. 3.—Thoracic 4.

FIG. 4.—Thoracic 7.

have invaded the whole of the gray matter on the posterior and left lateral aspects of the central canal.

At the level of the twelfth thoracic, the hæmorrhage had assumed the form of an oval ring, which entirely separated the base of the posterior horn and a portion of the left posterior column from the remainder of the spinal cord. Within this ring of extravasated blood lay fibres which stained blue with the Weigert-Pal stain.

At the level of the first lumbar, the hæmorrhage occupied about the same extent, but within its limits the tissue was more profoundly disintegrated.

At the level of the second lumbar, the hæmorrhage had destroyed the whole of the left posterior horn and a portion of the left posterior column. No definite signs of growth could be seen at this level.

From this point downwards, the cord showed no signs of a focal lesion.

Distribution of the systemic degeneration. [Plate 3.]—At the level of the seventh thoracic, staining by Busch's modification of Marchi's osmic acid method showed degeneration in the whole extent of the posterior columns on both sides. At this level, the extreme upper end of the focal lesion could be seen as a hæmorrhage (Fig. 1) in the left posterior horn. But in spite of the apparently unilateral position of the focal lesion, both posterior columns were profoundly and equally degenerated.

At the level of the fourth thoracic, the root-zones and greater part of the postero-external columns were seen to be free from degeneration, which occupied mainly the postero-internal columns on both sides; but the cornu-commissural tracts were degenerated.

At the level of the first thoracic, the degeneration did not extend into the postero-external columns, but, with this exception, occupied the whole of the posterior columns. The cornu-commissural tracts were slightly degenerated only. [Fig. 3.]

At the level of the fourth cervical, the degeneration occupied the postero-internal columns only, and mainly their dorsal portions; the degeneration in the more ventral portions was much less than in the lower segments. The cornu-commissural tract was not degenerated. [Fig. 4.]

Above the level of the seventh thoracic, both lateral bundles of Gowers and the direct cerebellar tract of both sides showed profound degeneration, slightly greater in the left than in the right half of the cord. This degeneration extended throughout the cervical region and is visible in Figs. 2, 3, and 4. [Plate 3.]

There was definite degeneration in the region of the spino-thal-

amicus of both sides, greater on the side (right) opposed to the main focal lesion.

Some degenerated fibres were also seen on the ventral surface of the cord, especially in that part traversed by the anterior roots.

Summary of Post-mortem Appearances.

The right lung was solid with septic broncho-pneumonia and a small abscess was found at its extreme base.

The right kidney was the seat of a malignant growth (hypernephroma), characterised by columnar cells and the presence of pigment due to extravasated blood.

Secondary growths were found in the kidney and in the spinal cord.

That in the spinal cord had caused hæmorrhage which had extended beyond the limits of the metastatic growth. This focal lesion extended from the seventh thoracic to the second lumbar segment, occupying mainly the left posterior horn and the adjacent parts of the posterior and lateral columns. At the level of the tenth thoracic, it reached its maximum extent, destroying the whole of the left posterior horn and a greater portion of the left lateral column.

Acute ascending degeneration was found in both posterior columns.

Both lateral bundles of Gowers, the direct cerebellar tract and apparently the spino-thalamicus, also showed signs of acute degeneration.

CASE 11.—CHARLES H.

A case of unilateral syringomyelia, which has been under observation from December, 1903.

Motion remains unaffected and he shows no wasting of any group of muscles.

The reflexes are still normal. He suffers from arthropathy of the right shoulder, which at one time gave rise to intense deep tenderness over a part of the analgesic limb.

Sensation to pain, heat and cold was at first lost over the whole of the right upper extremity and the upper part of the chest on the right side. This loss of sensation gradually spread on to the face and scalp. It has always been more extensive to heat and cold than to pain.

Tactile sensibility, the sense of passive position, tactile discrimination and tactile localisation have never been in any way affected.

Charles H., aged 30, first came under the care of Dr. Head at the London Hospital on December 7, 1903.

Eight years previously he was driving an omnibus; wishing to descend from the box, he missed the strap and fell, striking his right shoulder violently. He experienced "a feeling of numbness" and came to the London Hospital, but no serious injury was discovered. At the end of a week he returned to work.

Some considerable time afterwards, he noticed that his shoulder-joint "clicked" when he moved his arm, although it was entirely painless.

But, about six months before his admission in 1903, he began to suffer pain whenever his shoulder "clicked." The pain was situated not only in the shoulder, but also around the right ear.

Shortly before admission, the right arm and shoulder began to swell and "became set"; when he placed his hand into his pocket, he could not remove it again.

He had had no other illness, and denied all venereal disease. He had been married nine years and had two healthy children.

He has been under observation from December, 1903, until the present time, and we have repeatedly examined him together, and separately. We shall, therefore, give a summary of his condition, noting under each separate heading any change that may have occurred in the condition of the part, or function, of which we are treating.

Physical Condition.

He is a splendidly-built man with well-developed muscles. His heart and lungs are sound and he has shown no signs of visceral disease.

Throughout, he has been intelligent and unusually trustworthy to control-tests. His answers over normal parts have always been quick and accurate.

Motion.—Throughout the last three years, motor power has never been affected, except from the pain produced by moving the right arm. The grasps have remained equal and powerful, and no muscles have shown signs of wasting.

He walks well and can stand perfectly with his eyes closed. The legs are neither spastic, paretic, nor wasted.

Reflexes.—The knee-jerks and wrist-jerks have remained normal, and both plantar reflexes have given a flexor response throughout. No ankle-clonus was ever obtained.

Condition of the right shoulder-joint.—When he was admitted to the hospital in 1903, the right shoulder-joint was much swollen

and evidently contained fluid. A radiograph showed that the head of the humerus had been eroded, so that its surface was concave instead of convex; there was also some excessive formation of bone around the trochanters and articular surface of the scapula. The fluid threw a dark hazy shadow round the joint. On February 8, 1904, a small quantity of fluid was removed from the joint; it was of a pinkish-yellow colour and sterile on cultivation.

A considerable part of the swelling seemed to be due to muscular contraction, which varied from time to time.

At first, the joint was excessively painful, especially when

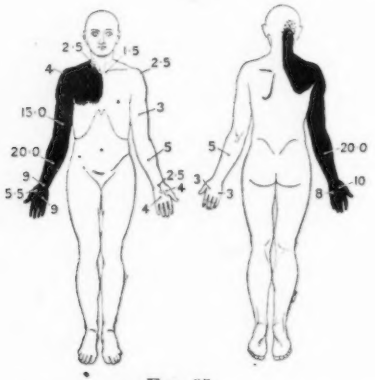


FIG. 55.

To show the loss of sensibility to pain in Case 11.

The area marked in black corresponds to the cutaneous analgesia. At various points are placed numbers corresponding to the pressure which caused pain. Thus 5·5 means that pain was caused by an algometer pressure of 5·5 kilograms. But such a number as 20 = 0 means that no pain could be caused even by a pressure of 20 kilograms.

the arm was moved. But gradually the pain subsided, and in September, 1906, movements produced no pain, although the joint was still swollen and measured 7 cm. more in circumference than the left shoulder.

Sensation.—(*a*) *Symptomatic changes in sensation.*—He has complained throughout of no abnormal sensation, except of pain in connection with the right shoulder-joint. This was aggravated by movement and relieved by keeping the arm at rest; although particularly severe during the hot summer of 1906, it had entirely disappeared by September of the same year.

(*b*) *Sensibility to pain*—(1) *Analgesia.*—When first seen, he was insensitive to the pain of a prick over the whole of the right upper limb, almost as high as the acromion. The cutaneous analgesia extended over the scapula behind, and over the third rib and third interspace in front.

This area has gradually increased under observation, and the extent of the cutaneous analgesia at the end of 1906 is represented on Fig. 55.

Throughout, the extent of the insensibility to the painful interrupted current has corresponded exactly with that to the pain of a prick.

In 1906, his sensibility to the pain of pressure was measured with the algometer, and the following readings were obtained :—

	R.	L.
Thenar eminence	Pain at 5·5	Pain at 4.
Centre of palm	Pain at 9	Pain at 4.
Ulnar palm over fifth metacarpal	Pain at 5	Pain at 4·5.
Dorsum of hand :—		
Over second metacarpal	Slight pain at 10	Pain at 3.
Over third metacarpal	Just painful at 11	Pain at 2.
Over fifth metacarpal	Pain at 8	Pain at 3.
Anterior aspect of wrist	Pain at 9	Pain at 2·5.
Forearm :—		
Flexor aspect	No pain at 20	Pain at 5.
Extensor aspect	No pain at 20	Pain at 5.
Arm :—		
Outer aspect	No pain at 15	Pain at 3.
Inner aspect	No pain at 15	Pain at 3·5.
Shoulder joint	Deep hyperalgesia (*vide* p. 702)	Pain at 2.
Acromion	Pain at 4	Pain at 2·5.
Sterno-clavicular joint	Pain at 2·5	Pain at 1·5.
Mastoid	Pain at 1·5	Pain at 1·5.
Temple	Pain at 1·5	Pain at 1·5.
Iliac crest	Pain at 2	Pain at 2.
Sole	Pain at 2	Pain at 2.

The results of these observations have been transferred to Fig. 55. The area marked in black is that which was insensitive to all cutaneous painful stimuli, and the numbers represent the pressure in kilograms which caused pain. It will be seen that over both the extensor and flexor aspect of the forearms and over the arm, pressure even up to 15 kilograms produced no pain. But no part of the hand was insensitive to this stimulus, although greater pressure was required to cause pain than over the normal hand.

(2) *Deep hyperalgesia.*—When first admitted in 1903, he

suffered intensely from pain in and around the right shoulder-joint. But, in addition to this pain, he was found to be tender to deep touch over an area which occupied, on the outer aspect of the arm, the whole of the region of the deltoid to a point somewhat distal to the insertion of that muscle. When touched with the head of a pin, or when the skin was pinched between the finger and thumb, this area was exquisitely tender, and by means of either of these manipulations, its limits could be mapped out with considerable accuracy.

There was no tenderness to light touches, but pain was pro-

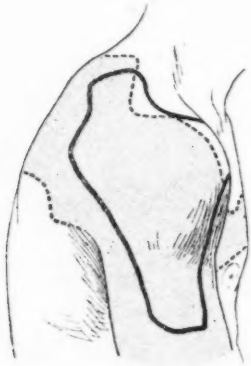


FIG. 56.

To show the relation of the deep hyperalgesia to the cutaneous analgesia in Case 11. The area of cutaneous analgesia is coloured grey and enclosed by a broken line; the area hyperalgesic even to the lightest pressure is enclosed by a heavy black line.

duced if the skin was tapped with cotton wool rolled into a hard wisp.

Yet the whole of this area, as far as it lay over the arm, was absolutely insensitive to the pain of a prick and to the painful interrupted current.

These observations were made and repeatedly confirmed during the first four months of 1904. Throughout the whole of this period the shoulder-joint was unusually painful.

A year later, in March, 1905, the joint was less painful and the area of tenderness less extensive. It then lay entirely within the limits of the cutaneous analgesia. If he was pricked within the

limits of this tenderness, he complained of an "acute sharp pain" which persisted for about a minute after the stimulus had ceased. But, when the skin over the same parts was gently lifted between the finger and thumb, the prick of a pin over the lifted skin was

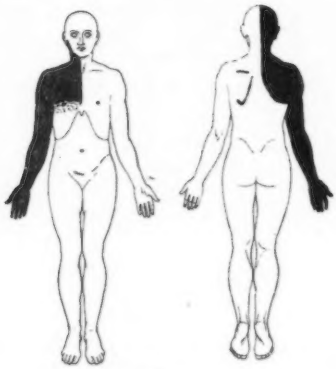


FIG. 57.

To show the loss of sensation to heat and cold in Case 11. The extent of the loss to heat on the face was occasionally somewhat greater than that of the loss to cold [*vide* Fig. 58].

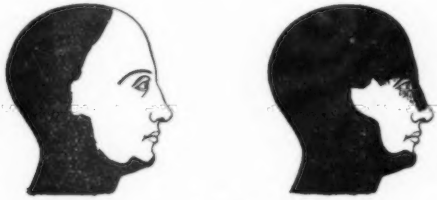


FIG. 58.

The extent of the loss to heat in Case 11 usually corresponded closely to that of the loss to cold. But at times the area insensitive to heat exceeded in extent that insensitive to cold, as shown in this Fig.

said to be a touch only; as soon as it was released, the prick was again said to cause pain. Any pressure over this area caused an "aching feeling."

In September, 1906, the joint was still swollen but no longer painful. All widespread hyperalgesia had disappeared. But, when tested with the algometer, he complained that any pressure pro-

duced pain. "Something seemed to shift in the joint which immediately caused pain"; yet the pressure was below 1 kilogram, and could not be registered on the instrument.

Here we were face to face with a true deep hyperalgesia, co-existing beneath an area of complete cutaneous analgesia. The lightest pressure stimuli caused pain and the extent of the hyperalgesia could be marked out on the skin. Yet the superficial layers of the skin were analgesic to the prick of a pin when raised from the subcutaneous structures.

As the cutaneous analgesia gradually advanced up the neck, the joint became less painful and the area of excessive tenderness disappeared.

(*c*) *Thermal sensibility.*—When he was first seen, the loss of sensibility to cold had not invaded the scalp, but occupied the right half of the neck only. Gradually, it assumed the form shown on Fig. 57.

The extent of the loss to heat usually corresponded closely to that of the loss to cold. But at times the two forms of sensibility were dissociated, as seen on Fig. 58, where the anæsthesia to heat exceeded that to cold. Over this dissociated area, he was carefully tested with temperatures of 25° C., 27° C., 35° C., and 40° C.; all were said to produce a sensation of touch only. None of these borders were at this time absolutely fixed, and the figures are given to illustrate the complete dissociation between the sensibility to heat and to cold, occurring over a part so sensitive to these stimuli as the forehead.

(*d*) *Tactile sensibility.*—Light touches were everywhere appreciated and there was no difference between the two upper limbs on careful testing with von Frey's hairs.

(*e*) *Acuæsthesia.*—He could distinguish the head from the point of the pin everywhere over the analgesic area of the right upper limb.

(*f*) *Appreciation of the difference in size* was tested by means of metal rods with diameters of 2 cm., 0·5 cm., and 0·2 cm. His answers were equally accurate over both upper limbs.

(*g*) *The vibrations of a tuning fork* (128) were appreciated everywhere equally on the two upper limbs. He was extremely quick in answering to a careful series of control experiments, which consisted of suddenly and unexpectedly damping the vibrations.

The legs were in every way normal.

(*h*) *The sense of passive position and movement* was everywhere preserved, even for movements of the grossly disorganised shoulder-joint.

(*i*) *The compass-test.*—Throughout, we have been unable to discover any difference in the compass-records obtained from the two upper limbs. His threshold (transverse) for the palms lies between 1 cm. and 2 cm.; at 2 cm. he has always answered correctly, and occasionally he has given a perfect series of answers at 1·5 cm. over both palms; but at 1 cm. he has uniformly failed to a varying degree.

(*j*) *Tactile localisation* was equally good over both arms and both legs.

Cranial nerves.—The pupils were equal, and reacted well to light and accommodation. The movements of the face, tongue, palate and vocal cords were perfectly performed.

Sphincters.—He had no trouble with the sphincters at any time.

Spine.—When first seen, his spine was straight; but by April, 1905, a slight degree of scoliosis had appeared, the concavity of the curve lying to the right, in the middle of the thoracic region.

CASE 12.—WALTER JOHN H.

First examined in March, 1904. *For eight years before this date he had been unable to appreciate heat and cold applied to the right hand, or to feel the pain of a suppurating finger. For one year the right half of the face had sweated excessively when he ate or drank.*

At this time there were no changes of any kind pointing to disease of the nervous system, except the loss of sensation to painful or thermal stimulation shown on Figs. 59 *and* 60. *His condition remained unchanged until September,* 1905.

By August, 1906, *the muscles of the right hand had become wasted and the hand had lost power. The loss of sensation had greatly increased, and had invaded the left hand* (*Figs.* 61 *and* 62). *Sensibility to light touch and pressure, and the sense of passive position and movement, were unaffected.*

Walter John H. was first examined in March, 1904. He was then 30 years of age and a fishmonger by trade.

He complained that for the last eight years or more he had noticed that the right arm was different from the left; it seemed colder and there was a sensation of tightness in his fingers. When he took a hot thing into his right hand, he did not at once recognise it was hot. In 1903, he poisoned the thumb of the

right hand; it "gathered" and suppurated, but he experienced no throbbing or pain, as with other fingers under similar circumstances.

For the last twelve months, it happened that whenever he ate or even at times when he drank, the whole right half of his face, forehead and head sweated profusely. He has been compelled to use a handkerchief to prevent the sweat running off his face.

Otherwise, he complained of no abnormal symptoms and said he was in the best of health.

He had suffered twice from gonorrhœa ten years before, but neither attack was followed by any symptoms pointing to syphilis. He was married, and had two healthy children.

His father died at the age of 47 of dementia paralytica, and his mother is highly neurotic; she has had several attacks of hysteria in which she has lost her speech. He has nine living brothers and sisters, all of whom are healthy. No collateral history of neuroses could be obtained.

From 1904 until April, 1905, the signs and symptoms of his disease remained unchanged, and we shall therefore give first the results of our examination in April, 1905.

Condition in April, 1905.

He was a largely built man, unduly corpulent for his age, but otherwise of healthy appearance. His pulse was regular, of good tension, and the arterial wall was not thickened. Heart and lungs were unaffected, and the urine was normal.

From the point of view of the nervous system, the only change was in sensation; there was no paralysis or wasting of muscles, no trophic changes in the skin or nails. His knee-jerks were equal and normal, ankle-clonus was not obtained, and both plantar reflexes gave a flexor response. The pupils reacted to light and accommodation. Vision was good; hearing, taste and smell were unaffected. No abnormality was found in the movements of the face, the tongue, the palate or the larynx. Both sphincters acted normally.

Sensation.—About 1896 he began to notice that whenever he had a cough a stabbing pain struck him in the right shoulder blade over a point at the vertebral border of the spine of the scapula. The centre of incidence of this pain gradually shifted upwards. At first it was situated close to the spine of the scapula; then it moved to the back of the neck, thence to the parietal region. From this point it moved forwards to the vertex, then to the temple, and in April, 1905, was situated

in the right eyeball. Each time he coughed he had a feeling as if he had been struck a violent blow in the eye.

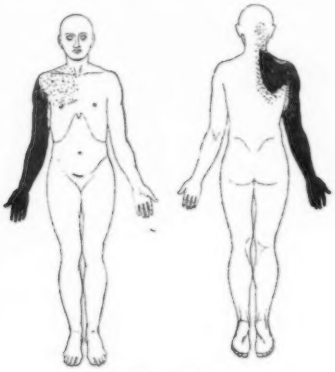


FIG. 59.
To show the loss of sensibility to cutaneous painful stimuli in 1905.

He complained of no abnormal sensations in the right arm and hand, except that in the winter they did not suffer from the cold weather in the same way as the left.

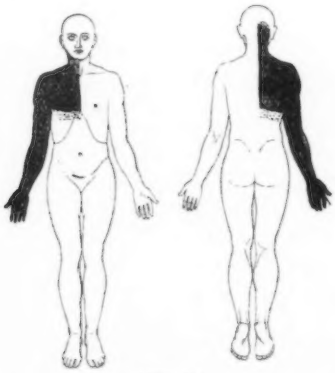


FIG. 60.
To show the loss of sensation to heat and cold in 1905.

Cutaneous painful stimuli were not appreciated when applied anywhere within the heavily-shaded area of Fig. 59. Over the parts dotted in this figure, a prick sometimes caused pain, but was frequently said to be a touch only.

He was entirely insensitive to all degrees of heat and cold over the darkened parts of Fig. 60. The border on the face and behind the ear was unusually definite.

The stimuli of light touch, deep touch and pressure were everywhere appreciated and accurately localised. He could tell the head from the point of a pin, even over parts that were completely analgesic. In the same way, he was able to discriminate everywhere between contact with a circular metal surface 1 cm. in diameter and a similar surface with a diameter of 2·5 cm. There was no loss of the sense of passive position or movement. The vibrations of a tuning-fork (128) were everywhere recognised. The two points of the compasses gave the same result on both the normal and abnormal hand: his answers were good, when the two points were separated for a distance of 1 cm., and applied transversely.

Condition in 1906.

In August, 1906, his condition had undergone a remarkable change. During the previous winter, he had noticed a gradual loss of power in the right arm, and the grip of the right hand had become less powerful. The little finger became gradually abducted from its fellows, when the hand was at rest. He also noticed involuntary twitchings in the muscles of the thumb and of the forearm.

He stated that in September, 1905, he experienced a sudden acute pain in the left arm, and since then had suffered from cramp in the left hand. These symptoms were, he believed, the same as those which preceded the loss of power in the right hand.

Motion.—The thenar and hypothenar eminences and the muscles in the first interosseous space of the right hand were definitely wasted. Fibrillary twitchings could be seen in the muscles of the right thumb, and in those which arise from the external condyle of the humerus.

The grip of the right hand was weaker than that of the left, and he had difficulty in opposing the thumb. All the interosseous movements were well performed.

The small thumb-muscles reacted slowly to the interrupted current, and, when the right abductor minimi digiti was stimulated, a certain number of fibrils only contracted, in contrast to the normal reaction of the synonymous muscles in the other hand. All the muscles reacted well to the constant current, except the

thenar group of the right hand, which reacted as easily with the anode as with the kathode.

The muscles of the left hand and arm were entirely unaffected.

Sensation.—A remarkable increase had occurred in the extent of the loss of sensation to painful and thermal stimulation (as shown on Figs. 61 and 62). The limits of the loss of sensation to the painful interrupted current corresponded exactly with those of the analgesia to prick. Over the whole of the thermo-anæsthetic area on the right arm, he failed to recognise heat and cold with any degree of temperature between melting ice and 60° C. Over the left arm the results were less definite, and he sometimes

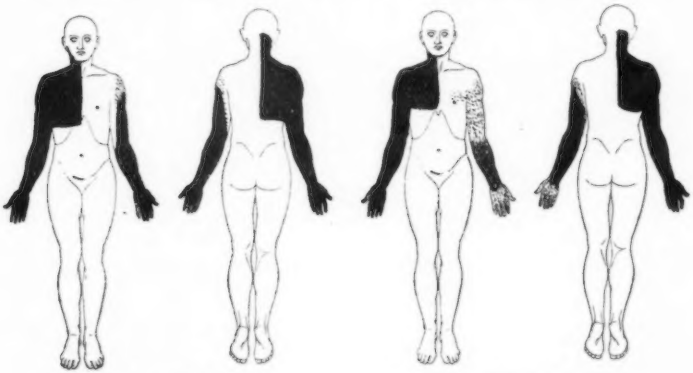


FIG. 61. FIG. 62.

To show the loss of sensation to cutaneous painful stimuli (Fig. 61) and to heat and cold (Fig. 62) in 1906. Evidently the loss of sensation was extending in the left upper extremity.

seemed to appreciate stimulation by the more extreme degrees of heat and cold, where water at 40° or 20° C. produced no sensation. Evidently, the loss of sensibility to heat and cold was here less complete than in the right arm.

The border on the face between parts that were sensitive and those that were insensitive to thermal stimuli was remarkably constant and varied little, according to the direction in which the tests were made. In 1905 the analgesia did not extend on to the face, as will be seen from Fig. 59. But, in 1906 the loss of sensation to painful stimuli spread until the residual sensibility was identical to prick, to heat, and to cold. Moreover, the border so marked out corresponded exactly with that deter-

mined by means of thermal stimuli nearly a year and a half before. This border is shown on Fig. 31, p. 612.

The algometer gave the following readings, which showed that deep pressure caused no pain over the whole of the right upper extremity. Over the left palm and flexor surface of the forearm, pain was not produced at first until the pressure was raised considerably above the normal threshold. But on repeating the experiment pain was produced by pressures no greater than those which are painful on a normal hand.

	R.	L.
Palm of hand	No pain at 10.	Pain at 10-7-4.
Over first metacarpo-phalangeal joint	No pain at 10.	Pain at 6.
Lower end of radius	No pain at 10.	Pain at 6.
Flexor aspect of forearm	No pain at 15.	Pain at 10-7-5.
Elbow	No pain at 10.	Pain at 5.
Outer aspect of shoulder	No pain at 15.	Pain at 5-3·5.
Sternoclavicular joint	No pain at 10.	Pain at 1·5-1.
Occiput	Pain at 1·5.	Pain at 1·5.
Temple	Pain at 2.	Pain at 2.
Over inner aspect of shin	Pain at 3.	Pain at 3.

The vibrations of the tuning-fork (128) were less clearly recognised over the whole of the right arm from the elbow to the shoulder-joint than over similar parts of the left side.

Stimulation with cotton wool and with pressure was everywhere appreciated, and the point of contact accurately localised.

No. 5 of von Frey's hairs (360 mgm.) produced a sensation of touch over both palms and the flexor aspect of both forearms.

The sense of passive movement and position was entirely unaffected, and there was no difference in the sensibility to the compass test between the two upper limbs. Everywhere, over both arms and both hands, he could tell the point from the head of a pin, even over parts that were insensitive to a prick, and he could appreciate the difference in size between metal discs of 2 cm., 1·4 cm., ·75 cm., ·2 cm.; but he was somewhat less accurate on the right side than the left.

Case 13.—Thomas B.

For three years, and probably longer, he had suffered from chronic disease of the spinal cord (syringomyelia).

Analgesia and thermo-anæsthesia existed over the areas in Fig. 63.

No other forms of sensation were affected.

The muscles of both hands, especially the right, were wasted.

The left elbow joint was disorganised.

The reflexes were unaffected.

Thomas B., aged 48, a labourer in a jam factory, came to the London Hospital on November 18, 1905, complaining of a painful swelling of the left elbow.

He dated his illness from a fall on to his head three years before. But, for more than three years, his hands had always "felt cold," even when he was sitting before a fire; and when he went to bed, it was more than an hour before they became warm.

During the last two years before he came to the hospital, he had noticed wasting of the muscles of his hands, first in the right and later in the left.

He had been in the Army, serving five years in India; there he had contracted syphilis, for which he was treated for a month only. Otherwise, his health had been excellent. He had been married for twenty-four years and his wife had had thirteen children.

Present Condition.

He has been under observation for over twelve months and his condition has changed little, if at all. He is a short but sturdily built man of fair intelligence. He was firm to control-tests, and, provided the experiments were not too complex, gave consistently accurate answers.

He was in every way normal, except for the following changes in the nervous system.

Motion.—The small muscles of the right hand were much wasted, the thenar and hypothenar eminences were flattened, and the thumb was extended, lying in the plane of the palm. All the interossei showed signs of atrophy. The proximal phalanges of the fingers were hyper-extended at the metacarpo-phalangeal joints, and the two terminal phalanges were flexed. No contraction could be seen or felt when he attempted to abduct or oppose the thumb; interosseous movements were impossible, and he could not abduct the index or little finger.

All movements of the wrist could be performed with ease, and the fore-arm muscles showed no signs of wasting.

The left hand was wasted, but to a less degree than the right, and the thenar eminence was flattened.

He could carry out all movements with his left hand and could even oppose and abduct his thumb.

None of the muscles of the left fore-arm were wasted, and all acted perfectly.

All the muscles of both upper extremities reacted well to the interrupted and to the constant current, excepting only the outer

thenar group and the interossei of the right hand; these muscles could not be made to react, even to a strong interrupted current. On stimulation with the constant current, they reacted slightly, more easily to the cathode than to the anode.

His gait was normal and he could stand steadily even with his eyes closed.

The muscles of the legs showed no signs of wasting or spasticity.

Reflexes.—The knee-jerks were in every way normal: no ankle-clonus could be obtained and the plantar reflex gave a flexor response on both sides.

Articular changes.—The left elbow was much swollen and dis-

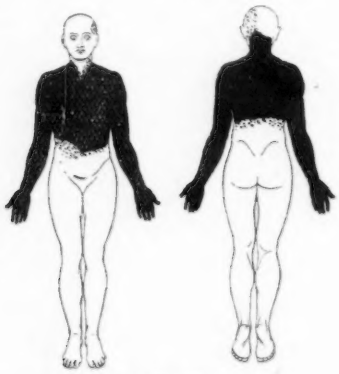


FIG. 63.

To show the loss of sensation to cutaneous painful stimuli and to heat in Case 13. The loss of sensation to cold is described in the text.

organised; creaking could be obtained on manipulation, and there was excess of fluid with much bony outgrowth around the joint. A radiograph showed much destruction of the opposing surfaces of the elbow-joint, with the formation of many osteophytic masses.

He complained of no other joint in his body, but a radiograph of the right elbow also showed slight bony changes.

Spinal column.—The spine showed a distinct lateral curve in the thoracic region with a convexity to the right, and a second curve towards the left in the region of the lower thoracic and lumbar spines.

Sensation.—He occasionally complained of pain in the left

elbow, probably induced by the nature of his work; for, owing to the loss of power in the right hand, he was compelled to use the left arm to raise heavy bags of sugar by means of a hand-winch. At no time could we elicit pain by manipulation of this joint.

The curious sensations of coldness in the hands, so marked a feature during the winter, greatly improved with the coming of summer. On an extremely hot day in September, 1906, these sensations had entirely disappeared.

(*a*) *Sensibility to painful stimuli.*—He was entirely analgesic to the prick of a pin and to the painful interrupted current over the area shown in Fig. 63; this was of somewhat greater extent in the winter than in the summer. But the borders of the loss of sensation always merged gradually into parts of normal sensibility.

The pain of deep pressure, as tested by the algometer, was completely lost over both upper extremities and over the upper part of the trunk. Elsewhere, pain was produced as usual, as will be seen from the following records. At each application he was able to recognise the gradually increasing pressure; but over the analgesic areas it caused no pain.

	R.	L.
Palm of hand	No pain at 15	No pain at 15.
Thenar eminence	No pain at 15	No pain at 15.
Elbow	No pain at 15	No pain at 15.
Shoulder	No pain at 15	No pain at 15.
Sternoclavicular joint	No pain at 10	No pain at 10.
Mastoid process	Pain at 3	Pain at 3.
Temple	Pain at 2	Pain at 2.
Sole of foot	Pain at 3	Pain at 4.
Inner aspect of tibia	Pain at 3·5	Pain at 3·5.
Outer aspect of anterior superior iliac spine	Pain at 5	Pain at 5.

(*b*) *Sensibility to thermal stimuli.*—During the winter, the thermo-anæsthesia corresponded almost completely with the extent of the cutaneous insensibility to pain; but during the summer of 1906, it was found that, although the insensibility to heat still corresponded with the loss of sensation to prick, the anæsthesia to ice was of somewhat smaller extent. A long series of experiments were made at this time over the outer aspect of the arms, where he seemed to be sensitive to ice, though insensitive to heat, and it was found that he was able to appreciate temperatures from 0° to 27° C., calling them all cold; but above this temperature, he seemed to be unable to appreciate certainly any thermal stimulus, calling 70° C. "a shade warm."

(*c*) *Tactile sensibility.*—The palms of both hands were horny

and insensitive; but, except over the hairless parts of the hands, he was normally sensitive everywhere to stimulations with cotton wool. Sensibility to pressure was nowhere disturbed.

(*d*) *Acuæsthesia and discrimination of difference in size.*—He could distinguish accurately the head from the point of a pin all over the analgesic area. Neither caused pain, but he described a prick as "like the point of a needle"; the head of the pin was said to be "blunter."

To test his power of recognising differences in size, steel rods were used with diameters of 2 cm., 0·4 cm. and 0·2 cm. His answers were invariably correct even over both palms.

(*e*) *Passive position and movement.*—To this test his answers were remarkably quick and accurate; even movements of the disorganised elbow were correctly appreciated.

When any muscle of one hand was thrown into action by stimulation with the interrupted current, he recognised the movement, imitating it perfectly with the opposite hand. Similarly, he could reproduce with the one upper extremity every movement produced in the other by electrical stimulation.

(*f*) *The compass-test.*—Over both palms the threshold to the compasses applied transversely lay between 2 and 3 cm. When applied longitudinally, he gave a perfect series of answers at 2·5 cm.

(*g*) Tactile localisation was nowhere affected, and we could find no difference between the two sides of the body.

Sphincters.—Micturition and defæcation were unaffected.

Case 14.—Florence R.

A case of syringomyelia, watched from 1899 *to the present time. The first signs consisted of wasting of the small muscles of the left hand with disturbed sensibility, which was transitory only.*

Gradually, the muscular wasting increased, and in 1902 *sensibility was again found to be disturbed in the left hand. This change progressed until, at the end of* 1906, *the cutaneous insensibility to pain was complete everywhere and the thermo-anæsthesia was widespread.*

No other form of sensibility has been disturbed at any time.

The reflexes and sphincters have remained unaffected.

Florence R. has been under observation since October, 1899 when she first came to the London Hospital, complaining of neuralgic pains in the occipital region and numbness in the left arm. For several months she had noticed a tendency to drop

things from her left hand but, until the muscular atrophy was pointed out to her at the hospital, she was not aware that the hand was wasted.

A stout, healthy looking woman of 24, she showed no abnormal signs except those pointing to disease of the nervous system.

The small muscles of the left hand were wasted, especially the interossei. The hand had begun to assume the "clawed" position, but the thumb lay in the normal position, and the thenar muscles, though flabby, were not definitely wasted.

At this time the only other abnormal sign consisted of a diminished sensibility to heat and cold over a small area on the forearm and hand (Fig. 64).

By January, 1900, the muscular atrophy in the left hand had

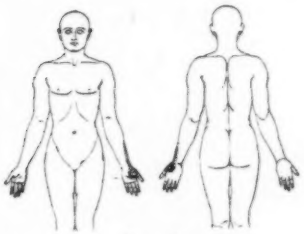


FIG. 64.

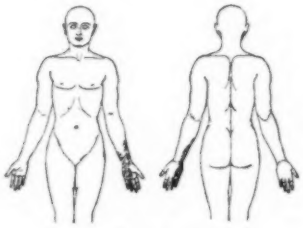


FIG. 65.

Fig. 64 shows the disturbance of thermal sensibility discovered in October, 1899. Sensation to prick was unaffected.

Fig. 65 shows the disturbance of sensibility to heat, to cold, and to prick, which appeared in March, 1902. For the intervening two and a half years no definite loss of sensation could be discovered in spite of the progressive muscular atrophy.

greatly increased, and the muscles of the left forearm showed signs of wasting, but no sensory disturbance could be discovered.

In March, 1900, the interossei, thenar and hypothenar muscles of the left hand were much wasted, and she could not oppose, abduct or adduct the thumb. The hand was "clawed," with the fingers over-extended at the metacarpo-phalangeal and flexed at the phalangeal joints. At this time the muscles of the right hand had begun to waste, more particularly the first dorsal interosseous and the muscles of the thenar eminence. The only muscles of either extremity which did not react normally to electrical stimulation were the interossei and the abductor and opponens pollicis of the left hand; none of these muscles responded to the interrupted current, but they contracted slowly to making the constant current more readily with the anode than with the cathode.

Throughout the first two and a half years, no definite change could be discovered in sensation except during October, 1899, when she first came to the hospital. But in March, 1902, after another severe attack of neuralgic pains, distinct loss of sensation to heat and cold and to prick was found over the area shown on Fig. 65. The muscular wasting had steadily progressed, not only in the left hand, but also in the small muscles of the right hand; the left forearm had already become affected.

In November, 1902, she suffered greatly from pain up the back of her neck and in both shoulder-blades, and was so unwell she was compelled to give up her employment. The loss of sensation advanced greatly in extent. Over the areas shown on Fig. 66, she was insensitive to stimulation with all degrees of cold; the loss of sensation to heat was somewhat more extensive on the left upper

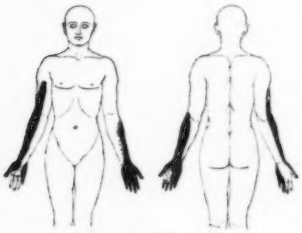


FIG. 66.

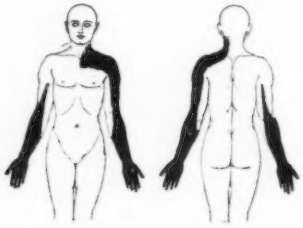


FIG. 67.

Fig. 66 shows the areas insensitive to cold in November, 1902.
Fig. 67 shows the areas insensitive to cold in June, 1903. The extent of the loss to heat and to prick is given in the text.

extremity, somewhat less extensive on the right. Sensibility to prick was nowhere lost, but was undoubtedly diminished over the left hand. Every other form of sensation was perfectly preserved.

The muscular wasting had made little or no progress, and the electrical reactions were identical with those obtained in March, 1900.

By June, 1903, the loss of sensation had progressed greatly. The greater part of the left arm and the whole of the right forearm and hand were insensitive to all degrees of cold (Fig. 67).

The extent of the loss of sensation to heat corresponded on the left arm with that to cold, but was less extensive over the right upper extremity. Analgesia to prick was almost confined to the left half of the neck and left upper extremity, where it corresponded exactly with the area on Fig. 67. Every other form of sensibility was perfectly preserved.

The muscular wasting had progressed very little, if at all, and the reflexes were unaffected.

The loss of sensation spread rapidly, and by November, 1903, had invaded the face and greater part of the scalp on the left side. Over the whole left half of the body, the loss of sensation was coterminous to prick, to heat and to cold (Fig. 68). But on the right side, although the thermo-anæsthesia occupied a considerable portion of the right upper extremity, analgesia to prick was found over the hand and part of the forearm only.

The muscles originally affected had continued to waste, but no further ones had been attacked since March, 1902. The thenar muscles and abductor minimi digiti of the left hand did not react to the interrupted current, but a response could be obtained from all the interossei in spite of their profound wasting. Every muscle of the left hand reacted to the constant current, even

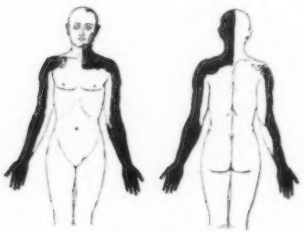


FIG. 68.

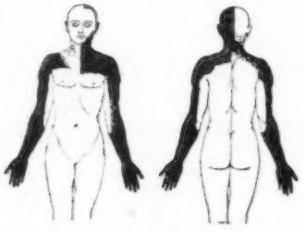


FIG. 69.

Fig. 68 shows the loss to sensation to cold and to heat in November, 1903. Fig. 69 shows how little the loss of sensation had advanced by February, 1904.

the wasted strips which represented the muscles of the thumb. In the right hand, normal reactions were everywhere obtained.

From November, 1903, to February, 1904, the loss of sensation did not extend on the left half of the body and head, but slight advance took place over the right shoulder and neck.

During this period, advantage was taken of the stationary condition of the symptoms to work out the extent of residual sensibility on the left half of the face (Fig. 33, p. 612). The border between normal and abnormal parts was found to differ little, whether the stimulus was a prick, or test-tube containing water at different temperatures.

By February, 1905, the loss of sensation to all three stimuli had extended considerably on the trunk, and the thermo-anæsthesia exceeded somewhat the loss to prick, and was no longer bounded by definite borders (Fig. 70).

The muscular wasting was at this time profound in all the small muscles of both hands, but greater in the left than the right. She could not make any movement with the small muscles of the left thumb, and adduction and abduction of the fingers was impossible. All movements of the right hand could be performed, but those requiring contraction of the intrinsic muscles were feeble. The only muscles which did not react to the interrupted current were those of the left thumb.

Condition from May to September, 1906.

She was a short, red-cheeked woman, of healthy appearance, except for her wasted hands.

Her speech is unaffected and she has never suffered from fits or sudden attacks of weakness in any limb.

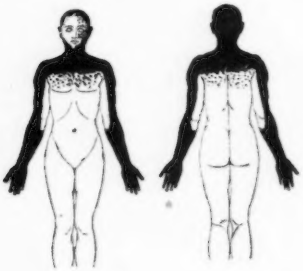


FIG. 70.

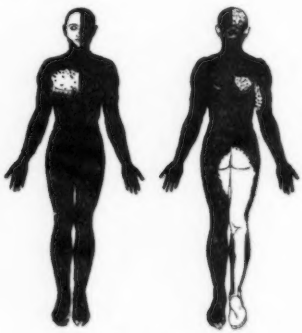


FIG. 71.

Fig. 70 shows the thermo-anæsthesia in March, 1905.
Fig. 71 shows the loss of sensation to cold in September, 1906.

Sensation.—She has suffered from much pain in the head, beginning in the left temple and passing over the forehead and bridge of the nose. The pain then passed into the spine, extending as low as the lumbar region.

Not uncommonly she experienced a curious sensation in the left half of the face, exactly as if it had been rubbed with menthol. Occasionally she has felt as if she had no left ear, and is compelled to touch it to assure herself of its presence.

During the extremely hot weather she was comfortably cool.

(*a*) *Sensibility to pain.*—The cutaneous analgesia had advanced

to such an extent, that there was no part of the body or face where she could appreciate the prick of a pin, or the painful aspect of an interrupted current.

Over both upper extremities and over both thighs no pain could be produced by excessive pressure, and the following records were obtained on the algometer :—

	R.	L.
Palm	No pain at 20	No pain at 20.
Forearm	No pain at 15	No pain at 15.
Arm	No pain at 15	No pain at 15.
Shoulder	No pain at 15	No pain at 15.
Sternoclavicular joint	Slight pain at 5	Slight pain at 5.
Temple	Slight pain at 5	Slight pain at 5.
Sole	No pain at 20	Pain at 5.
Outer malleolus	Pain at 10	Pain at 4.
Inner aspect of shin	Pain at 9	Pain at 3.
Knee	No pain at 15	No pain at 15.
Thigh	No pain at 15	No pain at 15.
Outer aspect of ant. sup. spine of ileum	No pain at 15	No pain at 15.

(*b*) *Thermal sensibility.*—She was insensitive to all degrees of cold stimulation over the areas shown on Fig. 71. But, over the parts which remain undarkened on this figure, she did not always respond, even to stimulation with ice. It was evident that the sensibility of these parts varied with her general condition.

To heat, especially the more extreme degrees, she was more widely sensitive; but it is extremely difficult to be certain how far this is based on a true appreciation of the nature of the stimulus, on account of her tendency to call even neutral touches "warm."

(*c*) *Tactile sensibility.* — Stimulation with cotton wool was everywhere appreciated, and she recognised No. 5 of von Frey's hairs over both palms.

(*d*) *Acuæsthesia.*—She could tell the head from the point of a pin everywhere, in spite of the total cutaneous analgesia.

(*e*) *The vibrations of a tuning-fork* were quickly recognised everywhere.

(*f*) *Sense of passive position and movement* was everywhere perfectly preserved.

(*g*) *Tactile discrimination.*—Over both palms the compass-records were perfect at 2 cm., when the points were applied longitudinally.

(*h*) *Tactile localisation* was good everywhere; a series of tests were made over the hands, using No. 5 of von Frey's hairs as the stimulus, and she named the spot touched with remarkable accuracy.

Motion.—The wasting and loss of power had not changed materially from the condition in 1905.

Reflexes.—The knee-jerks were normal. No ankle-clonus could be obtained and both plantar reflexes gave a flexor response.

Sphincters.—She had no trouble with micturition or defæcation.

Cranial nerves.—The pupils were equal and reacted normally. Movements of the face and tongue were well performed.

On ophthalmoscopic examination, disc and fundus were found to be normal.

Case 15.—George B.

A case of chronic intramedullary growth or syringomyelia.

When first seen by us in October, 1906, he complained that sensation had been altered in his right hand for seven years, and that he had gradually lost the power of writing.

Profound loss of sensation was present over the right upper extremity. The loss of tactile sensibility was unilateral (Figs. 75 and 76), but to painful and thermal stimuli the left upper extremity was also affected (Figs. 72, 73, and 74).

The sense of passive position was absent in the fingers and thumb of the right hand. Tactile localisation and tactile discrimination were unaffected over parts which were sensitive to touch.

Early signs of wasting and some weakness were present in the right hand. The right leg tended to be spastic.

The right knee-jerk was greatly increased, and the right plantar reflex gave an extensor response.

Rotatory nystagmus was present in both eyes when he looked to the right.

No other abnormal signs were found in the territory of the cranial nerves.

For some time he had had difficulty in passing water and in holding his motions when they were loose.

George B., aged 35, a painter, came to the London Hospital in October, 1906, for numbness and loss of power in the right hand.

Seven years before, he began to notice that the hand seemed always cold. On many occasions he burnt himself and knew

nothing of it until he saw the burns on his right hand. He could retain an object so long as he saw what he was holding, but often dropped it from his right hand, ignorant that it was no longer in his grasp. The power of writing had gradually left him.

Two years before he came to the hospital his right hand was poisoned by the bite of a mosquito, but the inflammation and even the incisions which became necessary on account of the swelling were entirely painless.

For eighteen months he had noticed stiffness of the right hip with some "numbness" of the sole of the right foot, and this leg had "jumped" at night.

He had noticed some difficulty in passing water for a considerable time before coming to the hospital, and for ten months had lost all sexual desire.

A bony projection had been slowly growing at the distal end of the proximal phalanx of the right thumb.

He was temperate and denied infection with syphilis or gonorrhœa. He had been married for ten years; his wife had never been pregnant.

Condition from October, 1906, to February, 1907.

He was a stout, heavily-built man, somewhat slow of speech, but intelligent, and able to concentrate his attention during the testing of sensation.

Except for the symptoms and signs produced by the disease of the nervous system, he was in every way normal.

Motion.—He walked on a somewhat broad base, but did not drag either foot, and could stand well with the eyes closed. He could not stand on either foot alone. When he attempted to stand on the left foot with the right raised, a few swaying movements preceded his fall, but when he raised the left foot he fell instantly.

The right leg was held stiffly and, if he was examined after he had been walking about, all the joints could be moved passively without undue effort. But the right leg was distinctly spastic after he had been for some time in bed, especially at the end of a morning spent in sensory stimulation whereby the leg had been frequently thrown into spasmodic contraction. The right leg was not paralysed or wasted.

The condition of the left leg was in every way normal.

There was no wasting of either hand, but fibrillary twitching was noticed in the third and fourth interosseous spaces. The grasp of the right hand was somewhat more feeble than that of

the left (right 16 kgm., left 23 kgm.), and the fingers were out of alignment, coming together unsteadily. This was especially noticeable in the little and ring fingers; but every movement of the right hand was possible. The extensors of the right wrist acted less powerfully than those of the left forearm; every other movement of both upper extremities was well and equally performed. All muscles, including those of the right hand, reacted normally to both forms of electrical stimulation.

Reflexes.—The right knee-jerk was greatly increased compared with the left. No ankle-clonus could be obtained, but the right plantar reflex gave an extensor response. That on the left side was of the normal flexor type.

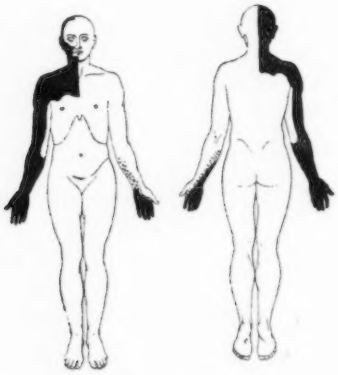


FIG. 72.

To show the loss of sensation to cutaneous painful stimuli in Case 15. The patch of remaining sensibility on the inner aspect of the right arm was worked out by testing from sensitive towards insensitive parts.

Sensation.—(*a*) *Sensibility to painful stimuli.*—The whole of the parts marked on Fig. 72 were insensitive to cutaneous painful stimuli. Over the inner aspect of the right arm, sensation was still present to prick and to the painful interrupted current, and the borders of this area in the long axis of the arm were sharply defined.

The condition of the left hand and forearm varied from day to day, but at the best sensibility to cutaneous painful stimuli was gravely diminished, and on several occasions the greater part of the hand was entirely analgesic.

The algometer gave the following remarkable results, which are the average records in kilograms of many examinations.

	R.	L.
Palm of the hand	Pain from 10 to 13	No pain with 15.
Dorsum of the hand	Pain at 7	Pain at from 4 to 7.
Flexor and extensor aspects of forearm	Pain usually absent at pressures below 13	Pain usually absent at pressures below 13.
Olecranon	No pain at 14	Pain at 6.
Shoulder joint	No pain at 13	Pain at 6.
Clavicle	Pain at 8	Pain at 3.
Angle of jaw	Pain at 3	Pain at 2.
Temple	Pain at 1·5	Pain at 1·5.

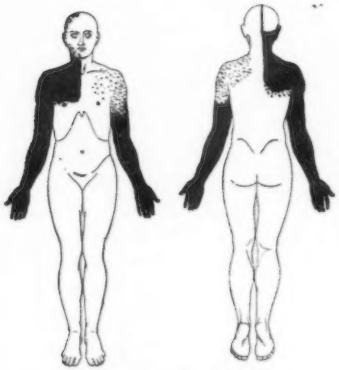


FIG. 73.

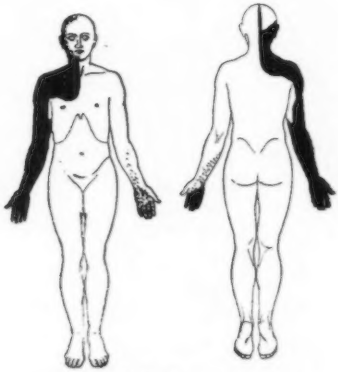


FIG. 74.

Fig. 73 shows the loss of sensation to heat in Case 15.
Fig. 74 shows the loss of sensation to cold in the same case. Here the patch of remaining sensibility on the inner aspect of the right arm was worked out by testing from sensitive towards insensitive parts.

Over the remainder of the body there was no loss of sensibility to painful pressure, and the algometer gave equal readings on the two lower extremities.

	R.	L.
Sole	Pain at 2	Pain at 2.
External malleolus	Pain at 2·5	Pain at 2.
Shin	Pain at 2	Pain at 2·5.
Inner aspect of knee	Pain at 4	Pain at 4.
Front of thigh	Pain at 5	Pain at 5·5.

It is evident from these records that the loss of sensibility to the painful aspect of the pressure was less extensive than that of the cutaneous analgesia. Moreover, the right hand still remained

sensitive to painful pressure though to a diminished degree, in spite of the complete insensibility of the whole forearm and hand to cutaneous painful stimulation.

(*b*) *Thermal sensibility.*—He was insensitive to all degrees of

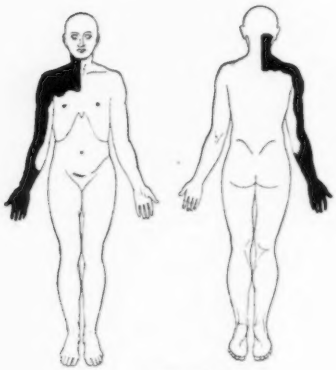


FIG. 75.

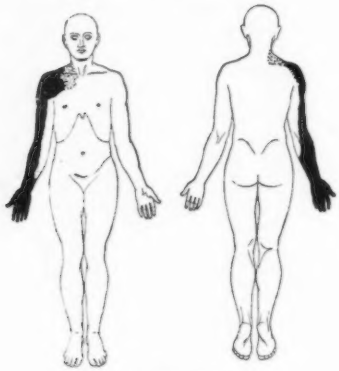


FIG. 76.

FIG. 77.

Fig. 75 shows the extent of the anæsthesia to cotton-wool in Case 15.
Fig. 76 shows the parts insensitive to deep touch in the same case.
Fig. 77 shows the relation of the borders at which different tactile stimuli no longer caused sensation. At the line marked "light touch" the patient ceased to appreciate cotton-wool and No. 8 of von Frey's hairs (830 mgm.). At the border of the dotted area pressure frequently failed to evoke a response. Finally, over the black area no tactile stimulus, even the firmest pressure, was appreciated as a touch.

heat over the whole of the right upper extremity (Fig. 73). On the left side, the extent of the loss of sensation varied somewhat from day to day, and merged gradually into parts of normal sensibility.

To cold the loss was less extensive and a small area remained sensitive on the inner aspect of the right arm (Fig. 74). Over the left hand and forearm, the distribution of the anæsthesia varied somewhat, corresponding to the evident advance of the lesion on this side of the nervous system.

(*c*) *Tactile sensibility* was abolished over the whole of the area shown on Fig. 76. The absolute loss of sensation to pressure-touch merged gradually on the neck into parts that were sensitive to this stimulus. But on the inner aspect of the forearm was a well-defined area of residual tactile sensibility.

When cotton-wool was used, the whole of the parts on Fig. 75 were found to be insensitive, including the right half of the neck to the line marked on Fig. 77.

The condition of the right half of the neck between these two lines was instructive; when tested with von Frey's hairs, it was found that even No. 8 (830 mgm.) could not be recognised, as soon as the border in the neighbourhood of the jaw was passed; and yet, over the left half of the neck he uniformly responded to No. 2 (100 mgm.) Tactile sensibility was sufficiently diminished at the line marked on the face to prevent a response to such stimuli as cotton-wool and von Frey's hairs. But No. 8 of these hairs is a pressure stimulus, and with peripheral lesions can be appreciated over parts of cutaneous insensibility. We concluded, therefore, that all forms of tactile sensibility were diminished at the line marked on Fig. 77. At this border, cotton-wool and von Frey's hairs ceased to cause a sensation. The anæsthesia gradually deepened until within the dotted area even a pressure-touch frequently failed to evoke a response. Finally, within the area marked black no tactile stimulus was appreciated.

(*d*) *Acuæsthesia.*—He could tell the head from the point of a pin everywhere except over those parts of the right upper extremity insensitive to light touch. Over the area sensitive to pressure but not to light touch, he was unable to distinguish the head from the point. But, wherever he was sensitive to light touch within the analgesic area, his power of distinguishing the head from the point was perfectly preserved.

(*e*) *Appreciation of differences in size* could not be tested over the right upper extremity. But over the left hand and arm his answers were correct to rods of a diameter of 2 cm., 0·75 cm. and 0·2 cm.

(*f*) *The vibrations of a tuning fork* could not be appreciated anywhere over the right upper extremity, but over the left arm and hand he everywhere answered correctly.

(*g*) *Sense of passive position and movement.*—He could not recognise any movement of the fingers or thumb of the right hand, and was liable to drop anything he was holding unless his eyes were fixed upon it. He entirely failed to appreciate any movement of the fingers produced by electrical stimulation. Movements at the wrist were not recognised until they had exceeded 20°; at the right elbow, until they had exceeded 10°; but movements of the right shoulder-joint were readily recognised.

All movements of the left upper extremity and of the legs were readily appreciated.

(*h*) *Compass-test.*—This test could not be applied to the right upper extremity, on account of the loss of tactile sensibility. Elsewhere the records were normal.

The records obtained by testing the area on the right half of the neck insensitive to light touch but sensitive to pressure, show well the effect produced upon the compass-test by a general diminution of tactile sensibility, produced by an intra-medullary lesion.

	R.	L.
3 cm.	1 | 10 R. 2 | 4 R. 6 W.	1 | 10 R. 2 | 10 R
10 cm.	1 | 10 R. 2 | 5 R. 1 Doubtful. 4 W.	

(*i*) *Tactile localisation.*—Wherever he was able to recognise a touch, whether it were caused by pressure, cotton-wool, or von Frey's hairs, he invariably named the spot accurately. When allowed to grope for the spot touched he was never more than 3 cm. away, and was remarkably quick and accurate, provided it lay over parts where tactile sensibility was preserved.

Cranial nerves.—The pupils were equal and reacted normally to light, to shade, and to accommodation. The eyes moved well in all directions, except that, when he looked to the right, a bilateral rotatory nystagmus appeared; the rotation occurred in a direction opposite to movement of the hands of a clock. No similar nystagmus was present when he looked to the left. No abnormal condition was found on ophthalmoscopic examination. All movements of the face and tongue were normally performed and the palate moved well.

Spine.—There was no lateral curve or other abnormality.

Sphincters.—For some time he had noticed that he could not retain his motions when they were loose and that he had some difficulty in starting micturition.

CASE 16.—ROBERT ARTHUR H.

In June, 1902, he woke to find the left hand and arm numb; he noticed that over the numb parts heat and cold produced a sensation different from that over the normal parts.

Nine months later his throat became affected.

Painless swelling of the left shoulder-joint was present.

First seen March, 1904. His condition has advanced little since that date.

The motor affection consists of paralysis of the left vocal cord and the left half of palate.

Reflexes.—Left knee-jerk exaggerated, left ankle-clonus, left extensor plantar reflex.

Sensation.—Profound loss of sensibility to prick (Figs. 78 and 79), to thermal stimuli (Figs. 80 and 81), to all forms of tactile stimulation (Fig. 82).

Appreciation of passive position and movement lost in the left arm and left leg below the knee.

Tactile discrimination lost in the left leg; cannot be tested in the left arm on account of the loss of tactile sensibility.

Tactile localisation, (1) by naming, equally good in both legs, (2) by groping, profoundly changed in the left leg; normal in the right leg.

Robert Arthur H., aged 33, a general provision dealer, was first seen by us in March, 1904, and since then has been under observation from time to time.

He was apparently in perfect health until one morning in June, 1902, when he found on rising that his left hand and arm were numb. At the same time he noticed that heat and cold produced a different effect upon the sensation of this arm.

Nine months later, he began to suffer from choking sensations and his voice became affected. About the same time he found that the left leg troubled him in walking; it seemed stiff and awkward, although it "could feel perfectly." He then began to suffer from sensations of giddiness, and his eyesight became confused.

It was not until October 14, 1903, that the right hand was affected; it became red and swollen, and he suffered from a violent shivering attack in which he vomited. This was said to be due to a small poisoned wound for which the doctor bound up the hand with the fingers flexed into the palm. So he remained for a month, and, when the bandages were taken off, his fingers had stiffened permanently.

About nine months later, the left shoulder-joint enlarged, but the swelling was accompanied by no pain.

As a boy, he had had an attack of typhoid fever, and on several occasions had suffered from "influenza," most severely in 1890.

He has been a life-long teetotaler.

No history could be obtained of venereal disease, nor according to his account is it likely that he could ever have been infected. He was married in 1899 and has one healthy child, born in 1905. His wife has never miscarried.

Condition on Examination (*November and December*, 1906).

In the following account we shall give the results of a series of examinations, extending over the last two months of 1906, alluding briefly when necessary to his condition on previous occasions.

He is a medium-sized, strongly-built man, of unusual intelligence. He answered well and did not fail with the control tests used.

Cranial nerves.—A remarkable nystagmus was present, not only with movement in all directions, but also when he looked in front with his eyes directed towards a distant point. When looking to the right or to the left, the nystagmus was purely horizontal and at no time was it truly rotatory. Otherwise, the eyes moved well, and there was no ocular paralysis. He had perfect muscular control of both eyelids. The face moved well both on emotional and voluntary excitation. The tongue was protruded in a straight line, but the right half seemed to be smaller than the left; this appeared to be due to a greater contraction of the muscles of the right half of the tongue, whilst the left half was flattened and flabby. The left half of the palate was paralysed, and on laryngoscopic examination, the left cord was seen to lie in the cadaveric position, completely motionless. The right vocal cord moved well, passing across the middle line to meet the left cord during phonation.

Motion.—There was no wasting of any muscle in the upper extremities, but the right hand was held in a half clenched position, and he was unable fully to extend the fingers in consequence of changes in the joints. The subcutaneous tissues were somewhat swollen, and the fixation of the fingers, combined with this swelling, accounted for the difference in the grasp of the two hands (right 8 kgm., left 17 kgm.).

But the grasp of the left hand was also defective, as shown by the low reading of the dynamometer; this was probably due, not to muscular weakness, but to the profound sensory changes in this hand.

For the left upper extremity was profoundly ataxic; he could touch his nose clumsily when his eyes were open, but as soon as they were closed, the difficulty became greatly increased. Even when allowed to look, he could scarcely bring the tips of the two forefingers together, and when his eyes were closed, this movement became impossible.

There was no ataxy of the right arm or hand.

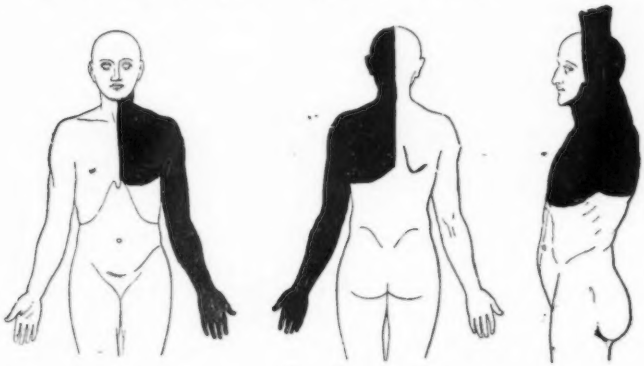


FIG. 78.

FIG. 79.

Figs. 78 and 79 show the loss of sensation to cutaneous painful stimuli in Case 16. The frontal border on the face (Fig. 79) was worked out by testing from sensitive towards insensitive parts. The caudal border shown on Fig. 78 was also worked out by determining the extent of residual sensibility to prick.

His gait was slightly unsteady, and on examination this was found to be due to the condition of the left leg. He could stand with his feet together, but when his eyes were closed, swayed and ultimately fell. In attempting to maintain his balance, the movements of the right foot were more extensive than those of the left; ultimately, he either fell towards the left, or thrust out his left leg laterally to act as a prop. Asked to stand on the

right leg only, he finally succeeded; but at first he had difficulty in maintaining his balance, owing to the absence of compensatory movements in the left leg, which was lifted. When he attempted to stand on the left leg only, he at once fell. Under such conditions, there was a striking difference between the violent balancing movements of the right foot and the complete quiescence of the left foot.

The left leg was slightly spastic, but was not paretic; nor were the muscles wasted.

All the muscles of the upper and lower extremities reacted perfectly to the interrupted and to the constant current.

Reflexes.—The left knee-jerk was greatly exaggerated, and ankle-clonus was obtained; the left plantar reflex gave an extensor response. On the right side the knee-jerk was obtained, but there was no ankle-clonus, and the plantar reflex was of the flexor type.

Sensation.—The loss of sensation was slightly less extensive in March, 1904, than in 1906. But since the changes which have occurred during the last two and a half years consist solely of a slight increase in the area affected, and not in the disturbance of any further sensory quality, we shall confine ourselves mainly to the present condition.

(*a*) *Sensibility to painful stimuli.*—He was insensitive to the prick of a pin over the whole of the left upper extremity, and over the left half of the neck and scalp (Fig. 78). The frontal border of the analgesia extended on to the face, including the left ear and part of the left cheek; the scalp was insensitive to a point a little in front of the vertical line joining the two ears (Fig. 79).

The lower border of the analgesic area on the trunk was remarkably well defined; it extended round the trunk, crossing the sixth, seventh, and eighth ribs in the axilla to end at the level of the sixth vertebral spine. When he was tested from parts of normal sensibility towards those that were analgesic, sensation to the prick of a pin or to a painful interrupted current ceased at the border on Fig. 78; from this, the border of returning sensibility scarcely differed by one centimetre, when we tested from insensitive towards sensitive parts.

Sensibility to a pin prick and to the painful interrupted current was perfect elsewhere over the body, except that it was somewhat diminished over the right forearm.

With the algometer, it was found that sensibility to the pain of excessive pressure was lost over the area insensitive to cutaneous painful stimuli. Over the right arm, the algometer

gave somewhat higher readings where cutaneous sensibility to pain was diminished.

	R.	L.
Temple	Pain at 2	Pain at 7.
Mastoid	Pain at 2	No pain at 10.
Sterno-clavicular joint	Pain at 3	No pain at 10.
Shoulder-joint	Pain at 6	No pain at 15.
Inner aspect of elbow	Pain at 6	No pain at 15.
Outer aspect of elbow	Pain at 7	No pain at 15.
Front of forearm	Pain at 6	No pain at 15.
Back of forearm	Pain at 10	No pain at 15.
Palm	Pain at 9	Slight pain at 15.
Back of hand	Pain at 5	Slight pain at 14.
Outer aspect of anterior superior spine of ileum	Pain at 3	Pain at 3.
Front of thigh	Pain at 4	Pain at 4.
Inner aspect of knee	Pain at 4	Pain at 4.
Shin	Pain at 2·5	Pain at 2.
Sole of foot	Pain at 3	Pain at 3·5.

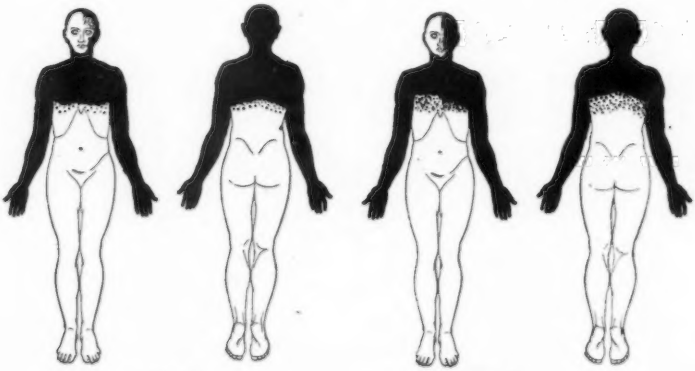


Fig. 80. Fig. 81.

Fig. 80 to show the extent of the loss of sensation to cold, and fig. 81 to show the loss to heat in Case 16.

(*b*) *Thermal sensibility.*—The area insensitive to all degrees of cold between 0° and 25° C. is shown on Fig. 80. The parts insensitive to all degrees of heat are marked on Fig. 81. These two figures almost correspond with one another except on the face. But, in 1904, the loss to cold considerably exceeded the loss to heat; the area insensitive to cold nearly corresponded with that on Fig. 80, but the extent of the anæsthesia to heat

more nearly agreed with the area that is now insensitive to painful stimuli (Fig. 78). Thus, at this time the whole of the right arm was sensitive to heat, but insensitive to cold.

(*c*) *Tactile sensibility.*—Stimulation with cotton wool was not appreciated over the whole of the left upper extremity, and the left half of the chest above the level of the nipple (Fig. 82). Elsewhere over the body and limbs, this stimulus was perfectly appreciated.

Firm pressure was not appreciated over the same area (Fig. 82) as that insensitive to light touch. This complete abolition of tactile sensibility over the left upper extremity was present when he was first examined in March, 1904.

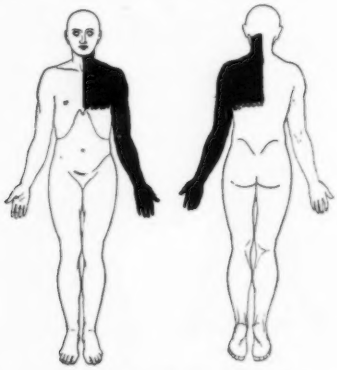


Fig. 82.

To show the loss of sensibility to stimulation with cotton-wool, with von Frey's hairs and with pressure-touch in Case 16.

(*d*) *Passive position and movement.*—He stated that he was able to appreciate the movements of the fingers of his left hand; but when they were moved passively he never recognised the movement, until the finger had travelled through an angle of over 60°. He was entirely unable to say into what position the fingers of the left hand had been placed. When the left thumb was tested, he answered as follows:—

Flexion	1 R. 9 W.
Extension	2 R. 8 W.

When allowed to look at the true position of his thumb, he volunteered that he thought it was pointing in a different direction.

At the left wrist-joint, he appreciated flexion and extension when movement had been made through an angle of 40°. He was, however, always correct in his appreciation of the position into which the hand had been placed by passive movements at the wrist, although his answers were somewhat slow.

Passive position and movement at the left elbow and shoulder were quickly and accurately appreciated; he recognised passive movement through 5° only.

In the whole of the right upper extremity, passive position and movement were accurately appreciated, and the difference between the condition of the right (normal) and left (affected) hands was most evident.

But it was in the lower extremities, where all other forms of sensibility were perfect, that the disturbed appreciation of passive position and movement was most striking. When his left great toe was moved, his answers were hesitating and very uncertain, but he quickly and accurately recognised all movements of the right great toe.

	Right.		Left.	
Great Toe	Extension	10 R.	Extension	4 R. 6 W.
	Flexion	10 R.	Flexion	9 R. 1 W.

He could not recognise movements of the left ankle, until it had been moved through more than 20°, whilst on the right side he answered correctly with movements of less than 5°.

	Right.		Left.	
Ankle	Dorsiflexion	10 R.	Dorsiflexion	3 R. 7 W.
	Plantar extension	10 R.	Plantar extension	6 R. 4 W.

At the knees and hips his answers were equally accurate on the two sides.

At a later date the following similar results were obtained:—

	Right.		Left.	
Great Toe	Extension	10 R.	Extension	10 W.
	Flexion	10 R.	Flexion	3 R. 7 W.

	Right.		Left.	
Ankle	Dorsiflexion	10 R.	Dorsiflexion	1 R. 9 W.
	Plantar extension	10 R.	Plantar extension	6 R. 4 W.

The same facts were illustrated when the movements of the foot were produced by electrical stimulation of the muscles. If the right foot was dorsiflexed by the application of an interrupted current to the anterior tibial group of muscles, or plantar extended by stimulating the calf, he answered quickly, repeating the move-

ment correctly in the same leg. The same result followed the outward movement produced by stimulation of the peronei.

But, when similar stimulation was applied to the muscles of the left leg, he was completely puzzled, and answered "the leg jumps," whatever movement resulted.

(*e*) *The compass-test.*—Owing to the loss of tactile sensibility in the left upper extremity it was impossible to compare the behaviour of the two hands towards the compass-test.

But over the lower extremities, where tactile, painful and thermal sensibility were perfect even to the finest tests, a remarkable difference was discovered in the behaviour of the two limbs. For over the right sole he made one mistake only when the points were separated for a distance of 2·5 cm., and applied transversely; over the left sole he gave eight false answers when the points were 6 cm. apart. Over the outer aspect of the left leg he gave perfect answers at 8 cm., but over the right leg 15 cm. was well below the threshold.

	Right.		Left.	
Sole:				
Compasses applied transversely	2·5 cm.	1 | 10 R. 2 | 9 R. 1 W.	6 cm.	1 | 10 R. 2 | 2 R. 8 W.
Compasses applied longitudinally	4 cm.	1 | 10 R. 2 | 10 R.	4 cm.	1 | 10 R. 2 | 10 W.
			8 cm.	1 | 10 R. 2 | 4 R. 6 W.
Outer aspect of leg . .	8 cm.	1 | 10 R. 2 | 10 R.	15 cm.	1 | 6 R. 4 W. 2 | 5 R. 5 W.

(*f*) *Tactile localisation.*—(1) Tested by the power of naming the part touched.

A number of points were chosen, such as the ball of the great toe, the centre of the sole of the foot, the heel, the "outer ankle" (external malleolus), "the inner part of the leg over the calf," "outer side, and just below the knee," the knee-cap, the "centre of the calf," the "middle of the front of the thigh." All these points when touched were named quickly and accurately; no difference could be discovered between the two lower extremities. Nor did he seem to be slower in naming a point touched on the lower extremities than when he was touched on the right hand or arm.

Then his eyes were closed and the legs were moved passively into a fresh position. This movement in the right leg was fully appreciated, but he was unable to tell into what position his left

foot and leg had been placed; yet, in spite of this difference, he named the spots touched on the two legs in their new position with equal accuracy.

(2) Tested by Spearman's modification of the groping method. A hole was cut in the centre of a piece of thin cardboard, on to which was fastened a sheet of white paper. The patient sat propped up in bed, with his legs stretched out in front of him. He was given a long soft-pointed pencil, and his eyes were closed. The cardboard was then held over his foot about 1 cm. from the skin, so that the hole lay over the centre of the dorsal surface. Through this hole he was touched, and he then attempted to touch the spot with the pencil used as a pointer. Now, since the cardboard intervened between his foot and the pointer, except at the small hole, his groping efforts were marked on the white paper, and the distance of the final mark from the centre of the hole gave the measure of his accuracy. A series of experiments were made with the centre hole over similar points on the dorsun of the right and left foot, on the outer aspect of the legs, and on the front of the thighs. Six tactile stimuli were given at each spot, and the numbers given below represent the average deviation at each point.

	R. (normal).	L. (affected).
Dorsum of the foot..	5·7 cm. ..	7 cm.
Outer aspect of leg	4·3 cm. ..	6·8 cm.
Front of thigh	2·5 cm. ..	4·3 cm.

The deviation was uniformly a little greater on the left than on the right leg, but the difference was within the limits of error of observation.

Another set of experiments was made as follows: The legs were allowed to lie stretched out in front of him and his eyes were closed. A series of observations were made exactly in the same way as those quoted above. Then his legs were moved passively, his eyes remaining constantly closed, and another set of observations made by touching the same spot on the dorsum of his foot. The results can be tabulated as follows:—

	R. (normal).	L. (affected).
Before movement	9·7 cm. ..	9 cm.
After movement	9·7 cm. ..	13 cm.

It will be seen that the deviation on the sound side remained the same, although the leg had been displaced passively; but on the affected foot the deviation increased 4 cm. after the foot had been moved.

(*g*) *Acuæsthesia.*—Under normal circumstances he never made

a mistake between stimulation with the head and the point of a pin over the right arm and hand, right half of the trunk, and both lower extremities. The left upper extremity was insensitive to touch and pain.

But the following observation will serve as an instance of the erroneous answers which may be given, if the patient is in any way uncomfortable. On one occasion at the end of a morning's examination this test was applied to the right arm. He gave curiously false answers as follows:

$$\text{Right palm } \frac{\text{Head} \mid \text{2 R. 8 W.}}{\text{Point} \mid \text{10 R.}}$$

$$\text{Right forearm } \frac{\text{Head} \mid \text{4 R. 6 W.}}{\text{Point} \mid \text{10 R.}}$$

He then said he wished to pass urine, which he did copiously. From that time he gave perfect answers everywhere over the right arm, just as on previous occasions.

(*h*) *Appreciation of difference in size.*— This was tested with three metal cylinders, 2 cm., 0·75 cm., and 0·4 cm. in diameter. Except over the left upper extremity his answers were correct in every instance.

Case 17.—Frank L.

On August 3, 1904, *he was injured by the fall of a skip which struck him in the back.*

On August 9, *the spine was opened in the region of the first, second and third lumbar vertebræ. No abnormality was discovered, showing that the condition was not due to injury of the cauda equina.*

He gradually recovered and, when seen by us in May, 1906, *showed some spasticity of the left leg, associated with analgesia and thermo-anæsthesia of the right leg* (*Fig.* 83).

Over this area, he was sensitive to the tactile aspect of pressure, but excessive pressure produced no pain.

All other forms of sensibility were perfectly preserved.

Frank L., aged 25, was admitted to the London Hospital on August 3, 1904, in consequence of an accident when working on the Rotherhithe tunnel. A skip of concrete fell, striking him in the lower thoracic and upper lumbar regions. He did not lose consciousness, but when pulled from under the skip, his legs dropped as if they were broken.

When admitted to the London Hospital, a hæmatoma was

present over the second, third and fourth lumbar spines, and he was much bruised about the body. The knee-jerks and plantar reflexes were absent, and both legs were completely paralysed.

He did not seem to be improving, and on August 9, 1904, Mr. Rigby operated upon the spine. He removed the laminæ of the first, second and third lumbar vertebræ. The dura mater was opened, and the cauda equina was exposed. No signs of rupture or bruising could be discovered anywhere. The wound was sewn up again and healed by first intention,

Power considerably improved in both legs, and he gained control to a great extent over micturition. When first examined by us, he had reached the following condition.

Condition in May, 1906.

He was a largely-built, intelligent man.

Motion.—When walking, he dragged the left leg like a man with hemiplegia, raising his pelvis with each step so that the left foot should clear the ground. He could stand on either leg, but could not support himself on the left leg only for more than a few seconds.

This leg was stiff, especially below the knee, and he complained that he could not move it as quickly and easily as the right. All movements at the left hip and knee were strongly performed, but at the left ankle he moved slowly, although with considerable force. He could dorsiflex the foot, and plantar extension was good.

All movements of the right leg were well performed and it was not stiff or spastic.

Reflexes.—Both knee-jerks were exaggerated, the left more so than the right. Ankle clonus was obtained on both sides and both plantar reflexes gave an extensor response.

Spine.—The scar of a well-healed wound extended from the eleventh thoracic to the fourth lumbar spine.

A radiograph of the dorsal region of the spinal column showed no obvious signs of fracture or other bony change.

Sphincters.—He could pass his water normally; but if he desired to micturate he was forced to do so at once, or the urine ran from him.

Defæcation was not affected in May, 1906.

Sensation. (*a*) *Tactile sensibility.*—He was sensitive everywhere to cotton-wool, and no difference in this respect could be discovered between the two legs. He recognised increasing pressure equally over both lower limbs.

(*b*) *Sensibility to pain.*—He was insensitive to all forms of cutaneous painful stimulation over the area shown in Fig. 83.

Deep pressure caused no pain over the sole of the foot, nor over both aspects of the right leg. But, over the front of the thigh, above the limits of the analgesia, pain was produced by a pressure of 5 kilograms on both sides.

	R.	L.
Outer aspect of leg	No pain at 16 ..	Pain at 5.
Inner aspect of leg over tibia	No pain at 10 ..	Pain at 4.
Front of thigh above the limits of the analgesia	Pain at 5. ..	Pain at 5.

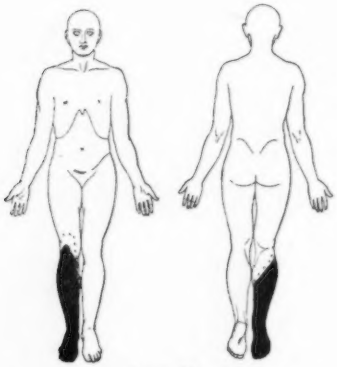


FIG. 83.

To show the loss of sensation in Case 17.

The dark area represents the parts insensitive to cutaneous painful stimuli and also to the pain of excessive pressure. It also corresponds to the extent of the thermo-anæsthesia.

(*c*) *Thermal sensibility* was lost for all degrees of heat and cold over the area marked in Fig. 83. The borders of this loss of sensation were ill-defined.

(*d*) *The vibrations of a tuning fork* (128) were appreciated with equal ease over both legs.

(*e*) The *sense of passive position and movement* was preserved everywhere.

(*f*) *The compass-test.*—The threshold over the outer aspect of the shins lay between 4 and 6 cm. when the compasses were applied longitudinally. We were unable to discover any difference between the two legs in their sensibility to this test.

(*g*) *Tactile localisation* was everywhere good.

Description of Plates.

Plate I.

Photographs of the spinal cord from Case 9 stained by the Weigert-Pal method. It will be seen that the whole of the left half was destroyed by a growth which occupied the centre of the cord. The white matter of the right half of the cord was distorted, but took the stain well.

Plate II.

Photographs of the spinal cord from Case 10 to show the focal lesion. Fig. 1 shows the sixth thoracic free from hæmorrhage or growth.

Plate III.

Photographs of the spinal cord from Case 10 stained by Busch's modification of the Marchi method. Fig. 1 shows both the extreme frontal extension of the focal lesion and the ascending degeneration. In the remaining figures all the ascending tracts are seen to be degenerated on both sides. (*Cf.* p. 697.)

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Reviews.

The Afferent Path.

The Afferent Nervous System from a New Aspect. By Head, Rivers and Sherren. Brain, 1905, vol. xxviii., pp. 99-115.

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Tipos Celulares de los Ganglios Sensitivos del Hombre y Mamierfos. Por Ramon y Cajal. *Trabajos del Laboratorio de Investigaciones Biologicas de Madrid,* 1906, pp. 1-28.

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The phenomena of sensation being more complex than those of motion, it is not surprising that in spite of much elaborate investigation and work devoted to its elucidation, our knowledge of the afferent path is lacking in many essential details, whilst many so-called facts concerning it are still uncertain.

For the purpose of this communication one may assume that the afferent path begins in the three sets of nervous mechanisms in the peripheral nerves, as summed up by Head, Rivers and Sherren, [1].

The Peripheral Sensibilities.

Head, Rivers and Sherren [1], and Head and Sherren [2], have recently shown in communications which add, not merely a page but an entirely new chapter to the science of neurology, that common sensation is based upon three kinds of sensibilities [1, p. 111], which are present and may be demonstrated in the peripheral nervous system, viz.:—

(1) Deep sensibility capable of answering to pressure and even capable of producing pain under the influence of excessive pressure. The fibres subserving this form of sensibility run mainly with the muscular nerves, and so are not destroyed by division of all the sensory nerves to the skin.

The peculiar aptitude, possessed by a part innervated *solely* by the afferent fibres of a muscular nerve [1, p. 103], is the appreciation of all stimuli which produce *deformation* of structure. Information is thus given of movement of joints, tendons and muscles, and also of the passive position of the same, and a very fair power of localisation is also rendered possible. Thus pressure or any jarring of the skin was sensibly appreciated in Head's case (complete cutaneous anæsthesia [1, p. 103]), and localised with remarkable accuracy. But when the hairs were pulled the elevation of the skin produced no effect upon consciousness. Pressure, which had previously caused a sensation, was no longer appreciated when applied to the skin lifted from the subcutaneous structure to form a ridge, and although pressure was localised with considerable accuracy all sense of form and size was lost over the parts affected.

(2) Protopathic sensibility capable of responding to painful cutaneous stimuli and to the extremes of heat (45° C. and over) and cold (20° C. and under). This belongs to the great reflex system, producing a widely diffused response unaccompanied by any definite appreciation of the locality of the spot stimulated.

(3) Epicritic sensibility, by which we gain the power of cutaneous localisation, of the appreciation of light touch, of the discrimination of two points (compass-test), and of the finer grades of temperatures (particularly from 25°-40° C., that is, those called warm or cool).

The above-mentioned sensibilities are often dissociated [1, p. 107] in an area affected by disease or operative procedure. According to the completeness of the lesion, the kind of nerves affected, and the state of repair, all forms of sensibility (superficial and deep) may be absent or more or less partially present. Thus an area may present protopathic sensibility and not epicritic sensibility, or even the epicritic and not the protopathic form. Moreover, the nerves subserving these two forms of sensibility do not coincide in their areas of distribution. For (*op. cit.* p. 110), provided the peripheral nerves are gathered into certain groups, it may be said that as regards light touch, and other forms of epicritic sensation, very little overlapping occurs, whereas in the case of protopathic sensibility enormous overlapping is found, and it becomes evident that whilst the unit of supply for epicritic sensibility, looked at broadly, lies in the peripheral nerves, the unit of protopathic supply lies in the posterior roots. Further, the two systems regenerate with unequal facility; for during the process of regeneration in a divided peripheral nerve in man, protopathic sensibility may begin to return in the subserved area in about 7 weeks (*op. cit.*, p. 163) (average 89 days), and be complete in 29 weeks (average 178 days). At this stage of the more primitive form (protopathic sensibility) there is the power to appreciate in the affected area pin pricks, extremes of heat and cold (above 45° C., and below 20° C.), but no power to appreciate or respond to light touches, small differences of temperature, and no accurate localisation is possible; in fact, fine discriminating power is absent. The return of protopathic sensibility brings a cessation of all those destructive nutritive changes that occur in parts where the skin is insensitive, such as ulcers, &c., which form as the consequence of burns or cuts, and do not heal so readily as on the normal skin. Such trophic changes are confined to parts deprived of protopathic sensibility. With the return of the latter ulcers and sores heal as readily as on the normal skin. Moreover, when a peripheral nerve to the hand is divided, it is noticeable that the palm begins to sweat at a time after union, which coincides approximately

with that of the return of protopathic sensibility. This sweating is innervated and controlled by the motor fibres of the sympathetic (the autonomic fibres of Langley and Anderson) that supply the skin.

As regeneration proceeds the higher and more discriminating form, viz., epicritic sensibility, begins to return in about 321 days after the lesion (*op. cit.*, p. 163), and though it varies somewhat, may be complete in about 364 days. Its return brings a power to respond to light touches, to localise accurately the sites of application of stimuli, and to appreciate correctly small grades and differences of temperature.

Course of Afferent Fibres to the Ganglia and Spinal Cord.

On their central path from the peripheral nerve mechanisms mentioned above, the afferent fibres mingle with efferent fibres to form the common trunks of the peripheral nerves. From these, bundles of both afferent and efferent fibres pass across between neighbouring nerve trunks; whilst in certain regions, such as the cervical, brachial, and lumbo-sacral, the intercommunications are very abundant and form the so-called plexuses. From the central side of them emerge the common nerve trunks on their way to the intervertebral foramina and the spinal cord.

In close proximity to the spinal cord the afferent fibres, as Bell and Magendie showed in the early years of last century, separate from the efferent to form the posterior or sensory roots, whilst the efferent fibres pass ventrally to form the anterior or motor roots.

Shortly after their separation from the efferent roots, the afferent fibres from the head and trunk enter the spinal ganglia, and it is now known that in these each fibre passes to a cell, namely, its cell of origin.

It has been proved, by the developmental and other methods, that these posterior or afferent fibres are merely prolongations of cells which, in the whole of the vertebrate series, start as bipolar cells. These during development—except in certain cerebral ganglia in all vertebrates, and in

the cerebro-spinal ganglia of fishes — are gradually transformed by the approximation and fusion of the bases of the two processes into unipolar cells. This solitary process then divides at a T-shaped junction, more or less at right angles, so that one branch passes to the periphery, whilst the other passes to the central nervous system. The peripheral branch terminates by ramifying freely in the skin, muscle, bone, serous membranes, viscera, &c., whilst the other terminates around cells in the central nervous system, as will be seen later.

Above the seventh thoracic segment the posterior roots on the central side of the ganglia pass from the intervertebral foramina horizontally to the dura mater of the spinal cord; below that level they pass inwards and more and more obliquely upwards according as they are more caudally placed. Further, in their transit from the dura mater to the spinal cord they pass obliquely upwards and inwards from the second cervical segment downwards, being more oblique as they are lower in the cord, so that the lowest lumbar and sacral posterior roots are actually vertically placed instead of horizontally, and thus run for a distance of some inches vertically in the spinal canal between the dura mater and the spinal cord. This simple anatomical arrangement, though of great importance, has not infrequently been overlooked in diagnosis, and thus led to serious errors in treatment.

The posterior (afferent or sensory) roots enter the spinal cord by many rootlets along the postero-lateral sulcus, whilst the anterior (efferent or motor) roots similarly enter the cord by many bundles of fibres along the so-called antero-lateral sulcus. The termination of the afferent fibres in the spinal cord will be given below.

Structure of the Cerebro-Spinal Ganglia.

The cerebro-spinal ganglia serve as the seats of origin for the afferent fibres. Hence the term exogenous for the latter, in contradistinction to those fibres called endogenous, which have their origin and termination in the central nervous system.

Needless to say, a great deal of work has been devoted to the morphological and physiological study of these structures, but so far the morphology is far in advance of the physiology, and, as may be gathered from this paper, many things in this connection remain hidden which the future will reveal.

The spinal ganglia are collections of nerve cells grouped more or less as islets on the course of strands of the posterior root fibres. The structure and arrangement of the cells have been minutely studied by a large number of observers, including Lenhossek [163], Held, Marinesco, R. A. Flemming, Cox, van Gehuchten, Nelis, Dogiel, Oloriz, Huber, Flesch, Orr and Rows, Warrington and Griffith, Ramon y Cajal, Nissl, Lugaro, &c. According to the results obtained by different methods of staining (chiefly, however, by the alkaline dyes, such as methylene or toluidin blue, Kronthal's method and Cajal's silver method) the cells of the spinal ganglia have been grouped according to certain types. Moreover, these types have been recognised not only in man but in the dog, cat, rabbit, and various mammals.

Among the cellular types are two distinguished by their well-marked characters and universal presence, which, according to the readiness with which they stain, have been called clear cells (*i.e.*, chromophobe) or obscure cells (chromophile). The chromophilic substance assumes various forms—it may be scattered throughout the protoplasm of the cell body in extremely fine granules or in irregular and coarse streaks, or even in lumps of variable size, from which fine filaments pass to neighbouring and similar structures, and thus give a reticular appearance. These chromophile granulations are embedded in the clear cells in a (chromophobe) non-staining substance, but in the obscure cells the fundamental substance stains deeply, and obscures, to a large extent, the chromophile substance.

Dogiel [3], after many years' study of this subject, has divided the nerve elements found in the spinal ganglia into five groups.

(1) The neurones of the first type, *i.e.*, the ordinary well-known afferent fibres with T junctions. These are of two

kinds, viz., large cell bodies with thick axons, and small cell bodies with fine axons.

(2) Cells which have one of their end-arborisations in the spinal ganglion itself instead of at the periphery in skin, muscle, &c.

(3) Cells with all their branches entirely included within the ganglion, and serving to couple up two or more of those mentioned in group 1.

(4) The "sympathetic" multi-polar cells. These are of unknown relations, except that they also appear to have the termination of their processes within the ganglion. Why they should be called "sympathetic" is not quite clear. (See also Bayliss [36], p. 193.)

On the other hand, Lugaro [4], working on several kinds of animals, namely, the rabbit, dog, cat, and guinea-pig, recognised the following five types of cells. To save time and repetition the character of the various types of cells in adult animals are summarised according to various observers in the following table.

With regard to the above it will be seen that Orr and Rows agree with Lugaro as to the distinctions of types I. and II., whereas Warrington and Griffith combine these two types into their type I. In each of types III. and IV. Lugaro is in agreement with Warrington and Griffith, but Orr and Rows divide each of these into sub-divisions "A" and "B." Again Warrington and Griffith discard type V. of Lugaro and Orr and Rows, but add another group which we may call type VI. It will be seen in addition that Warrington and Griffith have not only given the measurements of the cells of their various types, but also their proportional frequency in the ganglia of the second cervical, eleventh thoracic, and lowest lumbar segments of the cat.

Many of the observations cited above are incapable of interpretation in the present state of knowledge. Whether all the above types of cells are really distinct morphologically, still less physiologically, remains to be proved, and it is possible that widely different appearances are caused by different physiological (or pathological) states of otherwise identical cells. In this connection see Gustav Mann [8] and F. H. Scott [9].

TABLE.[1]

LUGARO [4]. Cat, dog, rabbit.	ORR AND ROWS [5] and [6]. Man.	WARRINGTON & GRIFFITH [7]. Cat.
Type I.—Large clear cells, largest in ganglion, and most numerous. Chromophile elements, granuliform, scattered throughout cell body, though at periphery slightly increased in size with clear zone (peripheral) around. Nucleus central and large, with perinuclear clear space	Same as Type I. of Lugaro. Most numerous of all cells	35μ-100μ. Peripheral zone increases after cut axone—Warrington, Flemming, Cassurer. Perinuclear zone usually, not always present.
Type II. — Clear cells, chiefly medium size, occasionally large. Next in frequency. *Fine* Chromophile elements, but around nucleus larger than above, and especially large at periphery of cell. Nucleus large, clear, central, with perinuclear and peripheral clear zone	Next most frequent. II. Distinguishable from I. by large size of peripheral chromophiles arranged in single ring	In ganglion of C. 2, 25·5 per cent.; Th. 11, 21·9 per cent.; L. 7, 31·5 per cent.; of all cells.
Type III.—Cells obscure or darkly staining. Many small, others medium size. Frequently irregular in shape. Chromophile elements very minute centrally, slightly larger towards periphery. Distinguished by fundamental substance being darkly stained. Nucleus dark, central, but relatively smaller than above. Perinuclear and peripheral zones clear, but less distinct than above	(*a*) Obscure cells. *Large* variety. Chromophiles larger and arranged in two concentric condensations, one around nucleus, one around periphery. (*b*) *Small* variety. Chromophiles very minute, irregular outline and scattered throughout cytoplasm. *In both.* Nucleus stains deeply, and is central. Peripheral clear zone well marked, perinuclear less	Described and figured in all papers since Flemming. Most numerous in ganglia from 10μ-56μ. In ganglion of C. 2, 68·1 per cent.; Th. 11, 72·6 per cent.; L. 7, 56·8 per cent.
Type IV. — Cells of small or medium size. Chromophiles *large and few* in number, regular in shape, with numerous branches. Achromatic substance unstained or only slightly coloured. Nucleus large, clear, central. Well-marked perinuclear and peripheral clear zones	As IV., but two varieties, : (*a*) Small size cell, but chromophiles smaller and more slender than in (*b*). (*b*) Medium-size cell, chromophiles larger and diamond or square shape, and not so concentrically disposed as in (*a*)	Cells coarsely granular, scanty. 35μ-50μ. C. 2, 4·3 per cent.; Th. 11, 2·5 per cent.; L. 7, 3·7 per cent.
Type V.—Cells of small or medium size, round or oval (vorticose cell). Chromophiles arranged concentrically around central point of cell, those at periphery large, cohering, elongated and united to each other by fine branches. Those centrally smaller and more irregular. Nucleus always excentric. Peripheral and perinuclear zones distinct	As in V. Chromophiles large, long and slender, and radiate from nucleus in successive rings wider apart further from nucleus. Nucleus excentric, separated by perinuclear band of chromophile substance.	Vorticose cell so rare—discard it. (Also Turner).
		Type VI. — Amongst smallest cells, some with less dense network and either fine or scanty largish granules. Fundamental protoplasm unstained. Nucleus proportionately very large and 1·9 per cent., 2·6 per cent., central 4·2 per cent.

[1] It will be remembered that in the cat and dog the 2nd cervical nerve supplies skin and small muscles; the 11th thoracic, skin, small muscles and viscera; and the 7th lumbar, skin and large skeletal muscles, but not viscera.

Structure of Cells in the Cerebro-Spinal Ganglia according to Ramon y Cajal [10] *and* [11].

Type I.—The well-known unipolar cell, *i.e.*, cell with one axis cylinder, which in its initial part is usually arranged as a glomerúlus.

This cell was first described by Flesch, and later by Dogiel, Oloriz, van Gehuchten, Lugaro, Retzius, Nissl, Holmgren [160], &c. It includes the clear and obscure cells given in the above tables (Types I., II. and III.). According to the above observers, on the body of each cell is a raised cone, which, though slightly fibrillated, is wanting in chromatic substance and gives origin to the axis cylinder. According to Cajal this unipolar sensory cell is the dominant form in man and mammals, and constitutes from 65 to 70 per cent. of the peripheral sensory neurons. In man it assumes exactly the shape and structure described by Dogiel, Oloriz [155] and Retzius in the cat, rabbit, &c. It has a strong axon which arises from a cone or from a depression in the cell body, and which is coiled up in a complicated fashion beneath the cell capsule to form what Cajal calls a glomerulus. In man also it is frequently noticed that this axis cylinder forms two or three loops beneath the cell capsule before emerging from the latter at a place more or less distant from its place of origin. Some cells exist in which the glomerulus itself is represented by a kind of pericellular orb (ovillo).

On the other hand, the glomerulus may assume a very simple form consisting barely of one or two cells. Finally, besides these glomerular cells, there are found, as in the cat, others which, as a rule, are much smaller and completely devoid of a corresponding structural arrangement.

Type II.—Multipolar cells, *i.e.*, cells furnished, in addition to the ordinary axon, with short stout dendrites ending within the capsule (see Figs. 1, 2, and 3).

Workers by the Golgi method such as Disse, Lenhossek, Spirlas, &c., have already described as present in batrachians, birds and some mammals, cells which possess, as well as the ordinary axon, dendrites terminating inside

FIG. 1.

A small cell of the ganglion of the vagus in man: *a*, axis-cylinder; *b*, short dendrites terminating within the cell capsule. (S. Ramon y Cajal.)

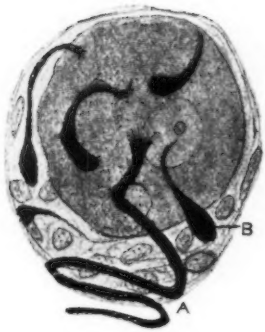


FIG. 2.

A cell of the cerebro-spinal ganglia of man: *A*, axon; *B*, dendrite terminating in a club-like swelling. (Ramon y Cajal.)

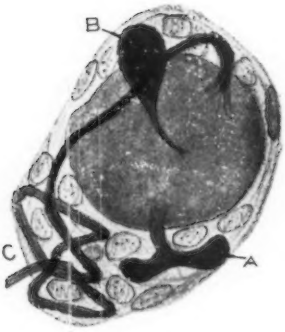


FIG. 3.

A cell of the plexiform ganglion of the human vagus; *A* and *B*, dendrites; *C*, axon. (Ramon y Cajal.)

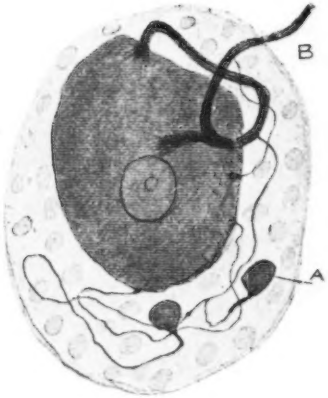


FIG. 4.

Cell with fine appendices terminating in sub-capsular bulbs (sheep).
(Ramon y Cajal.)

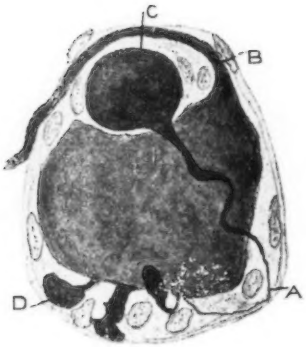


FIG. 5.

Human ganglionic cell possessing an appendix terminating in a large bulb, and various thick and short dendrites. (Ramon y Cajal.)

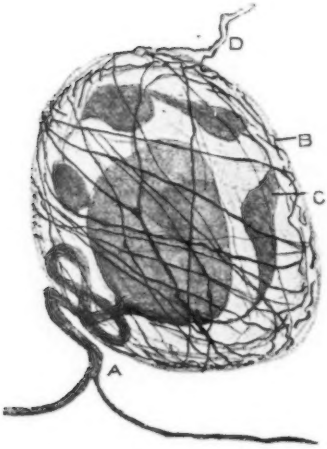


FIG. 8.

Ganglionic cell of man provided with thickened sub-capsular dendrites and a pericellular arborisation of nerve fibrils. (Ramon y Cajal.)

the cell capsule. This multipolar variety, according to Cajal, is common in man, dog, donkey, &c., and may be of small, medium, or large size. Its characteristic feature is the emission from different points of the cell contour of thick, short appendices (dendrites), which gradually increase in size as they pass onwards and terminate in a club or comma-shaped expansion beneath the cell capsule. With a good lens neuro-fibrils can be seen in each appendix. It is scarcely necessary to add that there are many variations in the number and exact form of the terminations of these dendrites. The axon starts and continues in the ordinary manner.

Type III.—Multipolar cells provided with fine processes or appendices which terminate in an encapsulated globe or bulb. Cajal has divided these into three varieties (*a*, *b*, *c*), (*a*) in which the bulbs are within the cell capsule, and these are most numerous (see Figs. 4 and 5); (*b*) those in which the appendices pass through the cell capsule and, with their terminal bulbs, end at a greater or less distance, even sometimes at the periphery of the spinal ganglion (see Fig. 6, *a*, *b*, *c*); (*c*) intermediate and transition forms, with bulbs both intra-capsular and extra-capsular.

According to the distinguished Madrid histologist, these cells (Type III.) are the most strange and unique elements yet known in the nerve centres. They are rare in mammals (cat, dog, horse), but common in man, particularly in the plexiform ganglion of the vagus (see Fig. 6). They were found, or at least a variety of them, many years before by Huber in an American frog. This isolated fact of Huber's had been unconfirmed until Cajal found to his surprise a number of these cells in the sensory ganglia.

Variety (*a*).—In which the encapsulated bulbs terminate within the cell capsule. This is the most numerous variety. The delicate appendices, always few in number, arise from the cell body or glomerular or other portion of the axon, and are made up of one or a group of neuro-fibrils. They increase gradually in diameter with distance from their origin, and terminate after describing one or two twists in a bulb or, occasionally, even in a row of bulbs (rosary) (see Fig. 8),

which usually lies against the cell body or in a depression of the same. The bulbs are homogeneous, stain darkly, and are occasionally unusually large. Between the cell capsule and the bulbs are a transparent substance and the subcapsular neuroglial cells (satellite cells).

Mixed forms are not wanting in which are present both fine threads with encapsulated globes, as well as thick and short dendrites.

Variety (*b*).—In which the appendices pass through the cell capsule, and end in their bulbs at a greater or less distance (Fig. 6). They are less frequent, and found only in man and certain mammals (horse, donkey, &c.). The appendices arise from the cell body or intra-capsular axon, or more frequently from the extra-capsular axon, and are very fine; and after various turns end in a large bulb within its special capsule. The latter reminds one in general appearance of Krause and Merkel's corpuscles; but their perfect homogeneity, absence of nuclei, and continuity with the fine dendrites easily distinguish them. These terminal bulbs lie occasionally at a considerable distance away from all cells, even in the white matter. Many of the appendices branch at right angles (see Fig. 6 *c*), and Cajal thinks it probable that all the long branches have a medullary sheath. Probably Dogiel's collaterals correspond to the branches of this type.

Variety (*c*).—Mixed forms with intra- and extra-capsular appendices are not rare. With slight exceptions they coincide exactly with the two previous varieties.

Type IV.—Fenestrated cells—found first by Cajal and Garcia [154] in the spinal ganglia of the dog and the ass (see Fig. 7.) The form and disposition of these cells vary infinitely in the same and different animals, but the most characteristic appearance is that in which the cell body, particularly at the region of origin of the axon, bristles with projections of the cell body, leaving cavities or fenestrations around which the neuro-fibrils, often hypertrophied, are arranged in cylindrical or irregular anastomoses. From many of these strings (or trabeculæ) the axis cylinder (which as a rule is thinner than they) emanates, and then passes in the usual way. These cells occur in health, but increase in number

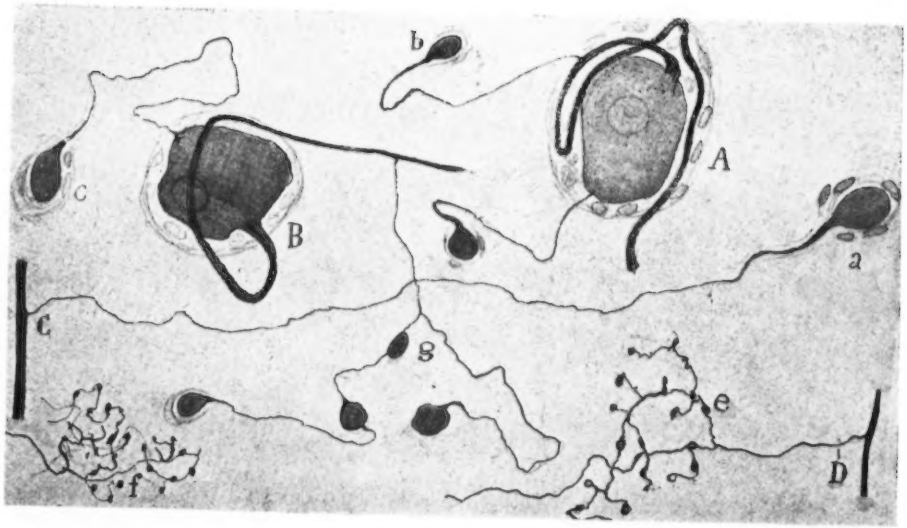


FIG. 6.

Showing various structures in the plexiform ganglion of the vagus of a young adult: *A* and *B* cells with appendices terminating in bulbs at some distance from the capsule; *C*, collateral from an axis-cylinder terminating in an encapsulated bulb. (Ramon y Cajal.)

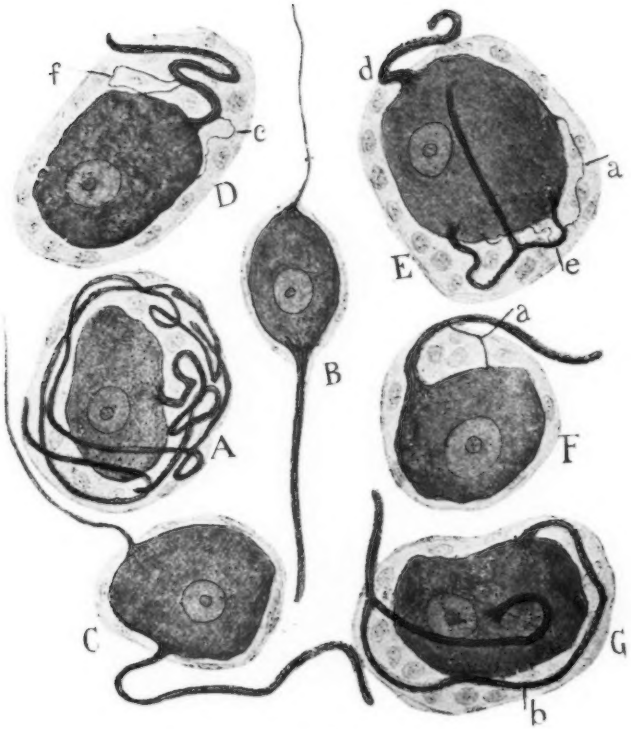


FIG. 7.

Different types of cells found in the plexiform ganglion of the human vagus: *A*, normal glomerular variety; *B* and *C*, bipolar cells; *D*, *E*, *F* and *G*, fenestrated cells. (Ramon y Cajal.)

with age and in pathological conditions, notably also under the influence of toxines (as in rabies and arsenical poisoning), and also from hyperplasia of the neuroglial cells (satellite cells) lying within the cell capsule. These cells have been observed in many animals, such as the horse, ox, pig, cat, lamb, &c., and in man. They occur in the root ganglia, and especially in the cranial ganglia, but are relatively less numerous and less complicated in man than in other animals. They have been found in the lamb at birth, but not in dogs under four months old. Cajal states the fenestration to some extent replaces, and is homologous with, the axon glomerulus, and serves to connect the cell more abundantly with afferent nerve arborisations. In his opinion not only the protoplasmic projections, but also the glomerulus, is induced or caused by the influence of the neuroglial satellite cells.

Type V.—In addition to the above definite types, Cajal shows the presence of decayed, degraded and senile cells, viz., usually small cells with a torn appearance of cell body, and surrounded by a large number of sub-capsular satellite cells.

Lugaro showed in the adult cat, and even in man, a small number of genuine bipolar elements (see Fig. 7, *b* and *c*) surrounded by a delicate capsule, and possessing a thick peripheral branch and a fine central branch.

Moreover, Cajal's observations reveal numerous nerve arborisations and pericellular nests similar to those described by Dogiel and Retzius in the cat (see Fig. 8).

The above observations have been confirmed, and in certain directions slightly amplified by Marinesco [12].

Remarks on the above Observations.

With regard to these striking discoveries of Cajal, one finds a great difficulty at first in bringing them into line with those of previous observers whose results are briefly referred to above. The physiological or other significance of some of his types of cells, particularly those of Type III., (viz., cells with appendices ending in encapsulated bulbs), present a problem difficult of solution. In this regard Cajal

states "No hay que disimular que las susodichas fibras globiformes plantean un problema dinamico de dificil solucion," and "Arduo seria, empero, en el estado actual de la ciencia, pretender adivinarlos." "De todos modos, la conjectura que surge en el animo cuando se medita sobra las posibilidades fisiologicas, es que los ganglos sensitivos, por excepcion de la regla general (los centros nerviosos son insensibles) estan provistos de aparatos cenestésicos ó sensitivos receptores, merced à los cuales pueden enviar à la célula, y después á la médula espinal, alguna excitación particular destinada á regular la inervación simpatica de los vasos sanguineos."

The view that the cells of Type III. act as sensory receptors, or receptive organs, has been combated by Nageotte [13], who points out their analogy, or even identity, with regenerating afferent fibres, as described by him more particularly in cases of tabes, and he states they are merely cells in process of replacing their axones.

Further, Cajal, in a personal communication just received, informs me he now holds a similar view. He considers that all the appendices provided with capsulated bulbs are newly-formed fibrils in process of becoming axones (at least in old age and in certain morbid conditions where the axones undergo destructive changes). This neo-formation is a normal process, for it is found even in young healthy adults and in animals, but is intensified enormously in advanced life, and especially in the ganglia of tabetics, as Nageotte has recently shown. He believes that this peculiar process is not always well regulated ("congruent"), but that it is often exaggerated (aberrant), and results from the presence of toxic substances in the reticulum, such as alcohol, morbid toxines, and other influences not yet determined. He bases his opinion upon the following facts, viz.: (1) The extraordinary resemblance which obtains between the appendices with bulbs and nerve fibres in process of regeneration. (2) The existence of these appendices in the white matter of the ganglion and even in the sensory nerve. (3) The presence of cells with bulbs in the cerebellum of dogs with distemper, and in rabid animals. (4) The enormous number of these cells in old animals.

RELATION OF GANGLIONIC CELLS TO AFFERENT FIBRES.

The cells are more numerous in the cervical than in the lumbar ganglia, and slightly least in the thoracic region, but remain constant in number through life. During this period, however (from early to late life), many small cells are gradually transformed into large cells (Hatai [42]), so that in the young animal the proportion of large to small cells would be 10 : 34, but in the adult 10 : 14. Moreover, not only would the average size of cells be increased, but slightly also the absolute size. (*Cf.* also Donaldson [43].)

The number of cells compared with the number of posterior root fibres for each segment, decreases relatively with age, but in the adult frog is about 2·45 or 3·26 to 1 (Hodge); 5 : 1 according to Bühler [161], and in the rabbit 6·4 : 1 according to Gaule and Lewin [51]. The posterior root fibres are more numerous in the cervical roots than in the lumbar, and slightly more in the lumbar than in the thoracic, and similarly they mature earliest in the cervical region and latest in the thoracic region.

As early as 1859 Stilling [45] stated that in each half of the human adult there are just over half a million posterior root fibres, and it is noteworthy that Stilling's and the above results are supported by a careful enumeration by Ingbert [46 and 161], who found in man on one side of the body just over 653,000 posterior root fibres (*i.e.*, 258,500 subserving the leg, 193,000 the arm, 117,600 the body, and 84,400 the neck). Of these, about a fifth (135,000) convey impressions from the muscles, and four-fifths (518,000) from the cutaneous structures.

These fibres increase both in number and size with age, being slightly more than twice as numerous in the adult rat as in the young [44]. According to Hardesty [47, 158] they diminish in number in the frog, with increased distance from the ganglia—this, however, is contradicted by Dale [48]. The fibres in the posterior roots are slightly fewer in number than in the corresponding peripheral branch [47, 159 and 44].

Further, the researches of Lugaro (working on the dog, rabbit and cat), of Warrington and Griffith (chiefly on the

cat), of Kleist (dog, rabbit and cat), and others, have shown that, after division of an afferent nerve, cells in the corresponding ganglion undergo morbid changes, which vary in rate and kind of alteration according to the types of cells affected. Yet although the chromophile elements may almost entirely disappear, complete restoration is possible; in other words, both in reaction and repair the cells are distinct morphologically and physiologically. Also the work of Orr and Lewis on the normal and pathological appearances of spinal ganglia cells in man are in agreement with the above.

As is well known, the afferent fibres from the viscera have their cells of origin in the spinal ganglia, and Warrington and Griffith have shown that by the Nissl method the axonal reaction after resection of the fibres is capable of indicating these cells (*op. cit.*, p. 314). They state that the proportion of cells in the spinal ganglia thus connected with the sympathetic system is very small, perhaps barely reaching at most 2 per cent., and judging from their experiments nearly all the cells belong to the small obscure cells. They are easily recognised from their chromatolytic changes, but do not belong to the smallest type of cell, since none of those measured were less than 30 μ.

Sherrington [51] first showed that in a muscular nerve trunk about two fifths of the myelinated fibres are from cells of the spinal ganglia, and that their diameters vary from 2 to 20 μ, and he finds that the diameter of the cells varies from 20 to 100 μ, or taking their volume as 1 : 125.

It has often been assumed that large fibres arise from large cells, and it was found by Warrington and Griffith that after section of a purely muscular nerve, namely, the hamstring nerve, chromatolysis occurred in certain large cells of the sixth, seventh, and eighth post-thoracic spinal ganglia.

Summarising their results (*op. cit.*, p. 324), they find that the large clear cell and the obscure cell give origin to the fibres distributed in the skin, viscera, and muscles. Of these the largest cells are exclusively connected with fibres from the muscle spindles. Large somatic and large splanchnic fibres have origin from cells of the same type, the large clear cell, small somatic and small splanchnic fibres also from cells of the same type as the obscure cell.

The coarsely granular cell is highly resistant to the axonal reaction, and may be a relay cell, but sufficient evidence for this is not yet forthcoming.

On Antidromic Nerve Impulses in the Afferent Nerve and Root Fibres.

Since the work of Goltz ([30] p. 184 *et seq.*), it has been known that the sciatic nerve contains fibres, excitation of which produces dilatation of the blood-vessels of the hind limb, and Stricker [31] in 1876 showed that these vaso-dilator fibres run in the posterior roots of the lowest two lumbar nerves. These results, denied by some, confirmed by others—Cossy [32], Gärtner [33], Morat [34], and Werzi-loff [35]—were not generally accepted, partly owing to their violation of the universally accepted Bell-Magendie law [36], but chiefly owing to possible fallacies in the methods employed. Bayliss has since shown in some remarkable work [36 and 37], that these vaso-dilator fibres of the afferent nerves and roots (which when stimulated produce vaso-dilatation independently of any possible concomitant diminution of tone in the vaso-constrictors) do not degenerate peripherally on division of the posterior root between the spinal ganglion and cord, but do degenerate on excision of the corresponding ganglia. Further, that even when a spinal ganglion is soaked in nicotine, vaso-dilator impulses pass undiminished to the periphery on stimulation of its posterior root, proving the absence of synapses (Langley [39 and 40]) in the fibres passing through the ganglion. He also proved that there are no vaso-dilator fibres to the hind limb in the abdominal sympathetic chain. Moreover, though Bayliss confirms Gaskell's discovery that the vaso-dilator fibres of the nervi erigentes run in the anterior roots of the second and third sacral nerves (it has been abundantly proved that vaso-constrictor impulses pass in the anterior roots), he finds that the only fibres to the hind limb acting as vaso-dilators are posterior root fibres excited antidromically, *i.e.*, the vaso-dilator action is conveyed along what are called afferent fibres in the contrary direction to that of the stream of afferent impulses. These

dilator fibres for the hind-limb (of the dog) run in the three lowest lumbar and first sacral roots, those for the fore-limb in the lowest three cervical and first thoracic roots, and those for the small intestine, not in the vagus, but in the lowest two thoracic nerves. It is of great interest that Bayliss and Head [41] have shown that these vaso-dilator fibres, after section in the cat, regenerate in about four to five weeks, that the return of their function is coincident with the return of protopathic sensibility and the disappearance of trophic lesions. Further, Bayliss states (p. 207 and p. 279) that the only hypothesis as yet put forward to explain all the facts of posterior root effects of vaso-dilatation is that the vaso-dilator fibres are identical with the ordinary sensory afferent posterior root fibres, according to which the same nerve terminations in the periphery serve both to take up sensory impressions and to convey efferent vaso-motor or vaso-inhibitory impulses to the structures in which they end.

In view, however, of some of the histological and other work of the last few years, it might have been safer merely to affirm that the vaso-dilator fibres of the posterior roots have the same trophic relations as those of the ordinary sensory or afferent fibres.

On Vascular Reflexes from the Afferent Path.

In this connection the well-known fact should not be overlooked that stimulation of a sensory nerve produces changes in blood pressure. This is even more obviously the case with excitation of a posterior root, and *à fortiori* of the posterior columns. In conjunction with Sir Victor Horsley, the writer has carried out during the last two years a series of experiments on dogs and monkeys to ascertain, more particularly by the plethysmographic method, the vascular reflexes produced on stimulation of the afferent path. The results will be brought forward later, but it may here be stated that stimulation of either a posterior root, the column of Burdach, or of Goll, the nucleus gracilis or cuneatus, or the ala cinerea in the monkey produces, when the vagi are intact, usually a fall of blood pressure, sometimes followed by a rise; whereas with the vagi divided (to prevent cardiac

inhibition) the effect has almost invariably been a considerable rise of arterial blood pressure.

Termination of Posterior Root Fibres in the Spinal Cord.

The fibres of the posterior roots do not all myelinate at the same period of development, and even in the adult are of various size and length and have different terminations in the spinal cord. Hence, though they all arise as outgrowths of cells in the spinal ganglia, they differ somewhat developmentally as well as anatomically and physiologically. Almost immediately after it enters the spinal cord each afferent fibre divides in the posterior columns into an ascending and descending branch, and from each of these again arise collaterals which pass horizontally to end in free ramifications in the grey matter. By various experimental and pathological processes it has been shown that each posterior root contains four or five main bundles of fibres, each of which terminates differently (Edinger [16]) (see also Fig. 9). One set of large fibres passes forwards in the grey matter to end around the cells of the anterior horn, a second set terminates by ramification around the Clarke-Stilling column of cells which is situated at the base of the posterior horn throughout the spinal cord, but chiefly developed between the upper lumbar and upper thoracic region. A third group, containing many large fibres, ends in little bundles of ramifications around the cells of the posterior horn, whilst of the remainder, though a few curve into the lateral column and rapidly end in the grey matter, by far the greatest number pass upward in the posterior columns of the same side, though none pass directly to the opposite half of the cord (Russell [14], Mott [15]). The total number of these, reinforced by incoming fibres above, increases from below to their termination around the cells of the posterior column nuclei in the medulla, but all the way some collaterals and main fibres are terminating in the grey matter. All those fibres which do not pass upwards (frontally) in the posterior columns soon end in the grey matter. Hence the afferent fibres which enter the

spinal cord have been divided into short (myelopetal) and long (bulbopetal), and a kind of anatomical sifting process exists, the functional significance of which will be pointed out later.

The descending branches (which help with fibres of different origin to form Schültze's comma tract) (Bastian [22], Schültze [23]) are generally short, and the majority of them merely extend through one or two spinal segments before ending like their collaterals in the grey matter. Various observers, however, have described marked variations in the extent of these fibres.[1]

The paths utilised in the spinal cord under normal conditions for the transmission of impulses from the periphery to the higher centres are those of least resistance, viz., the so-called long paths which may be briefly summarised here:—

The Posterior Columns.

The posterior columns, which in this connection are constituted by the uninterrupted exogenous fibres of the posterior roots. These fibres are made up of long, medium, and short fibres, most of which ascend vertically in the posterior columns, giving off along their course collaterals which terminate by division and subdivision around the cells of the grey matter, and more particularly the posterior horns. Each main fibre terminates in a similar manner to the collaterals, but the longest fibres find their end station in the medulla around cells of the nuclei of Goll and of Burdach. It is of importance that though the total number of fibres in the posterior columns steadily increases

[1] Thus Schäffer describes them as only extending through one segment. Dejerine and Theohari from the thoracic i. to thoracic iii., Margulies from thoracic vi. to viii., Wallenberg from thoracic iii. to v., but Marinesco from cervical vi. to thoracic x., Zappert from cervical viii. to thoracic viii., Marburg from the lower cervical to the sacral region, Collier and Buzzard ([59], p. 562) from the third lumbar to the coccygeal region, Risien Russell ([14], p. 164) from the cervical vii. to thoracic vi., v. Gehuchten ([62], p. 74) from cervical i. and ii., degeneration through three segments, &c. In the cat, dog and monkey similar differences in the records are given. In the cat Marinesco records one or two segments, Flatau in the dog eight segments, Margulies in the monkey from four to ten segments, Page May in the monkey from the upper cervical to lower lumbar region, &c.

from below up, the fibres in any given area diminish in number and some of them in size as they ascend, and there are many times more fibres entering the spinal cord than are continued up directly to the posterior column nuclei or indirectly through newly-formed long paths to the cerebrum. Moreover, the cells (column cells or Strangzellen) in the grey matter which give origin to fibres passing into the antero-lateral or posterior columns of the same side (*tautomère* cells of v. Gehuchten), or to fibres passing to the opposite white column (*hétéromère*), or even fibres which branch and pass partly to the same side and partly *via* the commissure to the opposite side (*hécatéromère*), must not be forgotten. These spino-spinal fibres (proprio-spinal Sherrington) serve to couple up various levels of the spinal cord, both above and below, and a certain number of these endogenous fibres are therefore found intermingled with the bulk of exogenous posterior root fibres. Hence in addition to ascending endogenous fibres[1] there are a certain small number of fibres which degenerate downwards and undoubtedly convey descending impulses of various kinds. [Bruce, Obersteiner, Flechsig, Hoche, Gombault et Philippe, Dejerine et Theohari, Page May.]

Singer and Münzer first showed, and it has since been frequently proved, that as the fibres ascend in the posterior columns they are displaced mesially by each incoming and higher (*i.e.*, more frontally placed) root. So that in the posterior columns of the cervical region a lamellar arrangement obtains (see Fig. 9), in that the fibres from the sacral region are placed mesially next the lumbar fibres, and most laterally the fibres of the inferior dorsal region, whilst the column of Burdach, which is distinguishable from about the fifth dorsal segment upwards, contains mesially the four upper dorsal fibres and more laterally the cervical fibres. Higher up (just above their termination) the latter arrangement is somewhat modified by the mesial fibres becoming more posterior and the lateral anterior (ventral). Further,

[1] Fibres arising and terminating in the spinal cord which undergo ascending degeneration, and apart from that have been proved to convey impulses upwards.

though in the past such weighty names as Schäffer, Thomas, Pelizzi, Sauques, Marinesco, von Sölder, Hoche, Quesnel, Tschermak, Dydynski, &c., have supported a contrary view, it may now be considered as certain (Mott, Sherrington, Tooth, Bruce [27], van Gehuchten [28]) that no posterior root fibres pass directly to the inferior cerebellar peduncle of the same (or other) side. Nor has it ever been satisfactorily proved in man or higher animals that posterior root fibres of one side pass into the posterior column of the other side (v. Gehuchten and many others), or to Gowers' tract, as stated by Rossolymo [52].

The Direct or Dorsal Spino-Cerebellar Tract.

The remaining ascending afferent paths in the spinal cord all belong to the central path, are constituted by fibres arising in a second neurone, and convey impulses which are transmitted to them from the primary peripheral neurone of the posterior root. These secondary sensory paths may be briefly summarised as follows:

The dorsal spino-cerebellar path or direct cerebellar tract path of Flechsig and Foville arises from the cells[1] of Clarke's column of its own side (Mott [24]). All these cells are provided with large and elaborate dendritic processes, and are imbedded in a complicated maze of fine fibrils coming from the posterior column. From these cells of Clarke arise almost uniformly large fibres, which sweep to the peripheral and posterior margin of the lateral column of the same side, and ascend in the cord immediately anterior (ventral) to the incoming posterior root fibres, and lying just external to the crossed pyramidal tract. They maintain this position till they reach the medulla, where they are joined by a large bundle of fibres from the crossed inferior olive, and then

[1] These cells are situated in islets at the base of the posterior horn throughout the cord, but more particularly from the first cervical segment to the second lumbar. They are of two kinds, namely, large cells of characteristic appearance, rich in chromophile substance, which is chiefly situated at the periphery of the cell, and smaller cells only poorly provided with small granules and chromophile substance. As only the first variety undergoes chromatolysis after section of Flechsig's tract it is supposed that they alone are the source of the fibres of that tract. (v. Gehuchten, p. 394; Sano.)

Homolateral impulses underlying muscular Sensibility—i.e. Sense of passive position and of movement, also of touch and pressure for a few segments.

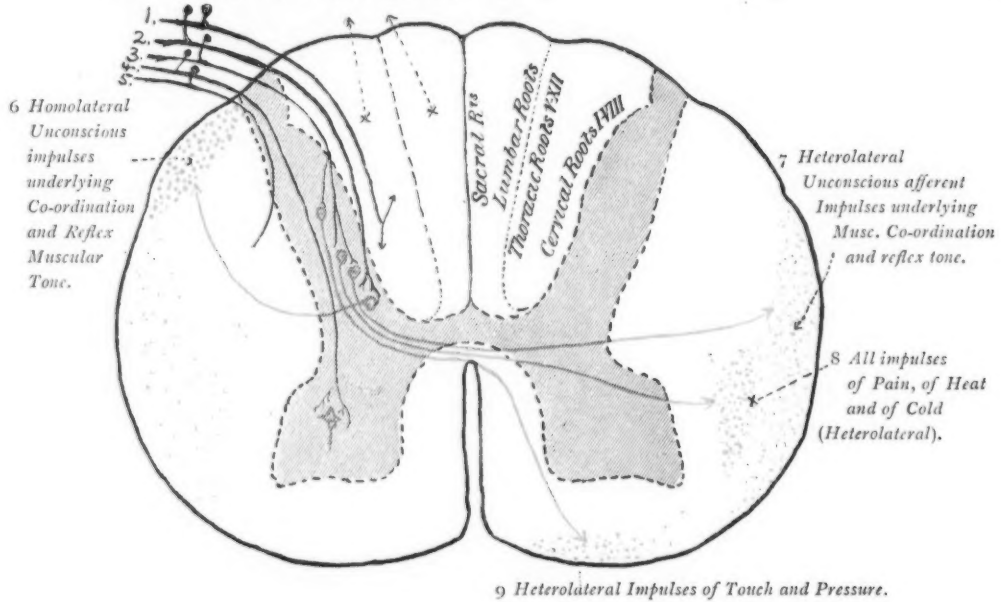

1.
2.
3.
4.
5.
6 Homolateral Unconscious impulses underlying Co-ordination and Reflex Muscular Tone.
Sacral Rts
Lumbar Roots
Thoracal Roots I-XII
Cervical Roots I-VIII
7 Heterolateral Unconscious afferent Impulses underlying Musc. Co-ordination and reflex tone.
8 All impulses of Pain, of Heat and of Cold (Heterolateral).
9 Heterolateral Impulses of Touch and Pressure.

FIG. 9.—Diagram to illustrate the termination of peripheral afferent fibres in the spinal cord, and the origin of the Secondary Central paths (partly after Edinger) with a brief summary of their function. 1. Bundles of fibres passing up in the posterior column—many myelopetal (to Sp. Cord) and the remainder bulbo-petal (to P. Col. Nuclei). 2. Fibres terminating around the cells of Clarke's Column. 3. Fibres arborising around cells in the Posterior Horn, and Intermediate Grey Matter. 4. Ditto around the Anterior Horn Cells. 5. Ditto swerving into the Lateral Column to neighbouring grey matter. 6. Direct, or dorsal Spino-cerebellar tract. 7 and 8. Gower's tract, *i.e.* (7) Ventral Spino-cerebellar tract. (8) Tract. Spino-thalamic et tectalis. 9. Ascending tract in the Anterior Column.—*W.P.M.*

pass with them latero-dorsally directly into the restiform body and thence to the cerebellum.

In their path in the spinal cord these direct cerebellar fibres are, to some extent, mingled with internuncial fibres which serve to couple up various levels of the spinal cord, and, by the degeneration method, it is clear that many of both kinds of fibres terminate in the grey matter as they ascend. Further, it is quite clear that both this direct cerebellar tract and Gowers' tract give numerous collaterals to the reticular formation in the upper cervical region and medulla, and also to the lateral nucleus of the medulla. (Thomas [171], Horsley and many others.) Dydynski [57] states that some fibres of the direct cerebellar tract pass to the posterior columns and terminate around cells of the nuclei of Goll and Burdach, but this version has not been accepted by the great majority of neurologists. Also the direct cerebellar fibres are successively displaced by the incoming higher fibres, so that a rough general lamellation obtains (Sherrington and Laslett [169]), in which the sacral fibres lie on the periphery of the spinal cord, the lumbar internal to them, the thoracic fibres still more internal, and the cervical fibres mesial to the others (see Fig. 9, Cajal and v. Lenhossek).

From the corpus restiforme and inferior cerebellar peduncle, the fibres of the direct cerebellar tract spread out in like manner, and terminate, according to Mott [18], in the dorsal vermis, according to Tooth, much as described by Mott. (See also Tooth's excellent figures in this journal, 1892, vol. xv., p. 399). Bruce described these fibres in 1898 as terminating in man in the lobus centralis, lobus monticuli, and slightly in the lobus lingualis of both sides of the vermis, but chiefly on the same side (homolateral). Edinger in 1904 (*op. cit.*, p. 217) described them as ending "almost exclusively in different portions of the vermis"; whereas v. Gehuchten, 1906 (*op. cit.*, p. 840), who has made a minute and detailed study of these fibres, describes them as ending in the posterior portion of the vermis. Hence, without multiplying descriptions, it will be seen that a great deal of uncertainty still exists as to the exact ter-

mination of the direct cerebellar tract in the cerebellum, though there can be no doubt that the superior vermis, especially its cortex, in whole or in part, is the chief end station of the direct cerebellar tract of Flechsig and Foville.

The Tract of Gowers.

The tract discovered by Gowers [25] in 1897 is a complex of fibres more or less intermingled, which, for the sake of clearness and simplicity, had now better be considered as constituted by the following divisions :—

(1) *The ventral spino-cerebellar tract* (often incorrectly called the tract of Gowers, of which it merely forms a part), arises in cells[1] of various sizes and form scattered throughout the grey matter (Ljubuschine [56], Laslett and Warrington [162]), chiefly of the posterior horn and intermediate grey matter of the opposite side, though slightly from cells of the same side.[2] The former pass along the anterior commissure to join their fellows of the opposite side, although some observers, relying largely upon data obtained in syringomyelia, consider that many of these fibres pass in the posterior grey commissure instead of the anterior white commissure. They consist in the main of finer fibres (see Fig. 10) than those of Flechsig's tract, and pass laterally to the peripheral margin of the lateral column [26], extending from the direct cerebellar tract behind to the level of the anterior roots in front.[3] This tract, like the direct cerebellar, increases in size as it ascends the spinal cord ; but instead of entering the inferior cerebellar peduncle, it continues directly on through the medulla oblongata lying superficially, and is mesio-ventral to the spinal root of the fifth. It passes latero-dorsal to the inferior olive, and is here mixed inextricably with fibres of the next tract (spino-thalamicus), and with Monakow's bundle (rubro-spinal) and with reticulo-spinal

[1] These cells are not exactly known. According to many observers they are in the posterior horns, though others, Rothmann, &c., say they are more ventrally placed ; and Lewandowsky, 1907, S. 187, even states that they arise from the Clarke-Stilling column of cells chiefly of the same side.

[2] Rothmann, *loc. cit.*, p. 47, says they arise chiefly on the same side.

[3] Gowers and Taylor, p. 201. The white matter is artificially divided into anterior and lateral columns, but there is no distinction of structure, and hence is often termed the antero-lateral column.

fibres. In the pons it becomes deeper, and lies between the spinal root of the fifth (which is postero-lateral), the facial nucleus (postero-internal), and the superior olive (mesial), and is broken up into little bundles by fibres of the trapezoid body. At this level a few fibres separate (Dydynski [57], v. Gehuchten [58], also Collier and Buzzard [59], v. Sölder [53]) to pass into the middle cerebellar peduncle of the same side, and thence into the cerebellum. The tract then passes posteriorly (dorsally) and hooks around (Mott) the root fibres of the fifth nerve to lie more and more superficially on the superior cerebellar peduncle. Here it forms a white band or several white strands on the surface, which in man may be easily seen by the naked eye. Thence the bulk of the fibres pass to the valve of Vieussens to end, according to Mott [18], in the ventral portion of the superior vermis, and Tooth [20], who is in agreement with Mott, gives figures of its termination.

These fibres of Gowers' tract end, according to Bruce [27], on both sides, but almost entirely homolaterally in the lingual lobule of the vermis, and according to v. Gehuchten (1906, p. 841), Loewenthal [17], Auerbach [19], Russell [21], in the anterior extremity of the superior vermis, and Edinger (1904, p. 217) states, "especially" in the frontal portion of the superior vermis. Lewandowsky, however, who holds a somewhat original view of their origin, states that they terminate ([61] p. 198) in the posterior portion of the vermis. Hence a great deal of uncertainty still exists not only as to the origin but also as to the precise termination of the main bundles of the ventral (and dorsal) spino-cerebellar tract. This is largely explained by the difficulty of limiting exactly a lesion to fibres of a tract which are almost inextricably mixed with those of other kinds, and thus an absolutely strictly pure degeneration of this one tract has rarely, if ever, been produced. The myelination and other methods have also so far not settled these points. The writer has a number of Marchi specimens made for various purposes, and showing degeneration in the spino-cerebellar tracts, but owing to the above difficulty they throw no further light on the question under consideration.

(2) *Tractus spino-thalamicus et tectalis.*—Further, in 1889 Edinger [67] showed in amphibia and lower vertebrates that out of the cells around which some posterior root fibres terminate arises a secondary path, the fibres of which pass ventro-mesially to the anterior white commissure, and thence across to the antero-lateral column of the opposite side, where they bend upwards to pass towards the brain. He believed these fibres to end in the thalamus, and this was shortly afterwards proved by Boyce [86] working by the experimental degeneration method in dogs. In 1901 Henneberg [66] found this tract degenerated in man, and described it as terminating in the most posterior and ventral portions of the lateral nucleus of the thalamus. A few months later Horsley and Thiele [63], in a case of fracture dislocation of the spine, traced these spino-thalamic fibres throughout their extent and stated that they end in the pulvinar of the thalamus, and constitute doubtless a tract of considerable phylogenetic importance. Further, the above and other observers have proved that these fibres, which pass to end in the (homolateral) thalamus, arise in conjunction with others (spino-tectal) which end in the mid-brain of the same side, but also slightly on the opposite side. At their origin and during their course the spino-thalamic and spino-tectal fibres are inextricably mixed, but lie, soon after they arise at the junction of the anterior and lateral columns, mesial (see Fig. 10) to the main bundles of Gowers' tract, and throughout their course become more or less commingled with the remaining fibres of Gowers' tract, though on the whole tend to lie mesially and differentiate themselves only towards their termination. Their course is excellently described and figured by Horsley and Thiele [63].

Moreover, the fibres of the spino-thalamic and tectal tract have widespread, if not abundant, connections, and a few of them send collaterals or terminal fibres to the lateral fillet of the same side (Mott, v. Monakow, Russell, Troschin [55], v. Sölder [53], Stewart [29], v. Gehuchten [58], and to the posterior corpora quadrigemina of the same and opposite side (Bruce [28], Loewenthal, Page May, Mott, Tooth, Stewart [29], Russell [14], Wallenberg [60]). A few fibres similarly

FIG. 10.

Photograph of a transverse section in the lower cervical region of a human spinal cord, which had suffered compression in the lower thoracic region. 1.—Column of Goll degenerated. 2.—Column of Burdach undegenerated. 3.—Dorsal cerebellar tract of Flechsig and Foville degenerated. 4.—Ventral cerebellar fibres and 5 spino-thalamic and tectal fibres of Gowers' tract degenerated. 6.—Ascending degenerated fibres in the anterior column.

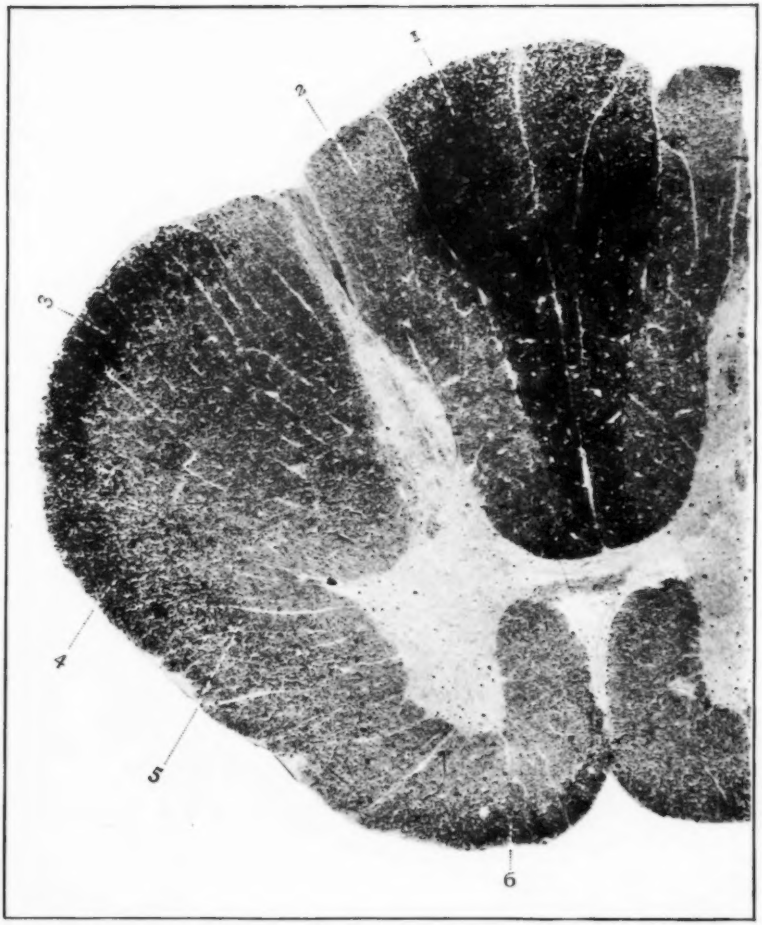


FIG. 10.

pass to the anterior corpora quadrigemina of both sides (Mott, Russell, Kohnstamm, Page May, Wallenberg [60], Thomas [61], but v. Gehuchten, p. 842, *contra*), and also a few fibres to the mesial fillet of the same side (Russell [21], v. Sölder [33], Troschin [55]), whilst a small, though larger, number of fibres terminate as mentioned above in the ventral and ventro-lateral portions of the optic thalamus of the same side. The statement of Morat [65] that "the tract of Gowers proceeds at all events partially to the cerebral cortex," certainly cannot be accepted in the light of recent work on the subject.

To sum up, Gowers' tract is a complex of fibres which arise chiefly (if not entirely) in the opposite half (Lewandowsky [64] *contra*) of the grey matter, and ascending in the antero-lateral column, sends a notable bundle of its fibres, *via* the superior cerebellar peduncle, into the superior vermis of the cerebellum. On its way it gives fibres and collaterals to the reticular formation of the cervical region of the cord, of the medulla and of the pons, to the lateral nucleus of the medulla, to the grey matter of (or surrounding) Deiter's nucleus, to the cerebellum, *via* the homolateral middle peduncle and direct sensory cerebellar tract of Edinger, to the lateral fillet and slightly to the mesial fillet, to anterior and posterior corpora quadrigemina of both sides, whilst the remaining fibres terminate in the ventral and ventro-lateral portion of the optic thalamus of the same side.

Ascending Path in the Anterior Column.

As Rothmann states ([67] p. 48), the conduction of afferent, impulses in the anterior columns of the spinal cord is of greater importance than has been hitherto considered. In these columns Edinger [69] has shown anatomically in fishes and amphibia, v. Wallenberg [70] in rabbits, v. Probst [71] and Rothmann [72] by the degeneration method in dogs, that out of the anterior columns fibres pass up directly to the optic thalamus. Lewandowsky (p. 203), however, denies the presence of such a direct path, and states that from the anterior columns only a chain of short ascending paths can be possible. Rothmann ([67] p. 48) reaffirms the presence

of direct fibres from the spinal cord to the thalamus, but adds it is possible they exist only from the higher cervical region upwards. Obersteiner (*op. cit.*, p. 410) considers that fibres probably pass from the anterior column into the middle fillet and thence to the optic thalamus.

On the other hand, fibres have been described by many observers which pass from the ground bundle of the anterior column up into the posterior longitudinal bundle, and thence to the middle line in front of the central grey matter and so to the cranial nuclei (Kölliker and Cajal), and even as high as the posterior commissure (Hoche and others). As in the other columns of the cord, ascending intersegmentary fibres (Bochenek, Sherrington) have been abundantly proved, and more particularly a bundle of fibres which lies in the area largely occupied by the descending tract of Marie, viz., along the angle of the anterior fissure [Troschin, Sherrington, Russell]. These fibres degenerate upwards, and, wholly or in part, have been termed the ascending tract of Marie. They constitute, doubtless, a complex of fibres, both crossed and uncrossed. Many of them arrive from the opposite half of the cord through the anterior commisure (Obersteiner, Dydynski, Ljubuschine, &c.), some of them are merely intersegmentary fibres (proprio-spinal, spino-spinal), and terminate in neighbouring grey matter, whilst the remainder are fibres which pass up into the posterior longitudinal bundle. Like better known tracts their exact origin and termination are obscure. Ljubuschine (*loc. cit.*) has traced them to the first cervical segment, and some of them terminate probably in the lateral nucleus of the medulla (Sherrington). Dydynski states that some of them pass up in the area of Helwig's descending tract to end in the inferior olive, and in this respect he confirms and amplifies Bechterew [73]. The writer has a number of specimens experimentally made in the monkey and dog, examination of which show ascending degenerated fibres which terminate in the inferior olive (see Fig. 11), and others which pass on to enter the mesial fillet just above, and internal to, the olive. Further, Dr. Gordon Holmes has shown me specimens of a case in man in which he has recently traced these spino-olivary fibres by the Marchi de-

FIG. 11.

1. Pyramidal Column. 2. Inferior Olive with numerous degenerated fibres terminating in it. 3. Recticular formation of Medulla.

generation method from the lower dorsal region distinctly into the inferior olive. A few of these fibres are also seen degenerated in the photograph (Fig. 10) of a section of the cervical spinal cord in a man who had suffered a nearly complete compression in the lower thoracic region.

Hence, therefore, apart from the long and short ascending intersegmentary fibres which constitute a chain-like series of shorter and longer connections in the anterior column, there are also fibres which come from the grey matter (chiefly posterior horns and intermediate grey matter) of the opposite half of the spinal cord, and ascend in this column to the important associational centre of the inferior olive. Here some of them terminate, whilst others exchange collateral branches, and are continued directly or indirectly to the optic thalamus. In addition to this there are a few direct fibres from the anterior column to the optic thalamus, as described in man and various animals by the observers mentioned above.

The Mesial Fillet or Medullo-Thalamic Path.

In the study of the fillets, the enormous inherent difficulties of the subject have been greatly increased by the contradictory terms employed by different observers for one and the same structure or part of a structure. Thus, to take merely one instance, the lateral portion in the pons and crus of what is now generally accepted as the mesial fillet,[1] has been variously called a part of the fillet layer, a part of the *Lemniscus de Henlé*, *Couche Rubanée*, *Ruban Cortical* or *Schleifenschicht*, or *Oberes Schleifenblatt*, or as partly or wholly synonymous with the *Obere Schleife*, *Hauptschleife*, and even the *Laterale Schleife*. Further, such terms as *Fuss der Schleife*, *Bündel vom Fuss zur Haube*, *Fusschleife*, *Bündel von der Schleife zum Fuss*, have occasionally increased the confusion (Forel, Reil, Burdach, Reichert, Meynert, v.

[1] The term mesial fillet in this communication is used in the sense now generally accepted, viz., to indicate the path from the posterior column nuclei to the thalamus (medullo-thalamic path), but of course includes within it certain fibres coming from cranial sensory nuclei, and a few others of unknown origin. It does not include the "lateral fillet," which is a tract made up chiefly of fibres from the cochlear nuclei, passing thence *via* the heterolateral superior olive (and nucleus of the lateral fillet) directly or indirectly to form a band in the side of the midbrain, which terminates in the nucleus of the posterior corpus quadrigeminum and in the posterior brachium.

Monakow, Bechterew, Flechsig, Schlesinger, Obersteiner, &c.).

It has already been mentioned that long exogenous fibres of the posterior roots and of the posterior columns terminate around cells of the nucleus gracilis and nucleus cuneatus. From these same cells arise fibres which course horizontally forwards as internal arcuate fibres to cross the raphe in the middle line, and then turn forward to form a compact bundle of fibres lying between the two inferior olives. The nucleus gracilis (which receives posterior root fibres from the fifth thoracic segment to the sacral region inclusive) is situated slightly more caudally than the nucleus cuneatus (which receives fibres from the cervical and four upper thoracic segments), and thus the internal arcuate fibres from the nucleus gracilis cross the middle line caudal to those coming from the nucleus cuneatus, and lie more ventral in the interolivary lemniscus (anterior) (Ferrier and Turner [74], Mott [82], v. Gehuchten [75], Bechterew [76], but Obersteiner, *op. cit.* p. 408, *contra*). It seems there can no longer be any doubt that *all* the fibres from the above nuclei decussate in the middle line and enter the interolivary layer of the opposite side (Singer and Münzer [77], Probst [78], v. Gehuchten [28], p. 827), though Vejas [79], Ferrier and Turner [74], Darkschewitsch and Freund have stated that some of these arcuate fibres remain on the same side and pass to the cerebellum *via* the inferior cerebellar peduncle. Obersteiner [*op. cit.*, p. 409], Tschermak [80], and Wallenberg [70] hold that some of these arcuate fibres are both homolateral and heterolateral, and pass from each side of the spinal cord to the inferior cerebellar peduncle of both sides.[1]

Arrived in the interolivary space they give abundant collaterals to the inferior olive (Held, Kölliker, Flechsig, Tschermak), and lying behind the pyramidal paths the fibres of the mesial fillet pass upwards (frontally) through the medulla into the pons, maintaining their relative position

[1] Probably the fibres that were seen to be degenerated by these observers had their origin not in the posterior column nuclei, but in the reticular formation or lateral nucleus of the medulla.

and increasing in bulk by accession of new fibres, some of which undoubtedly arise in the opposite sensory cranial nuclei (secondary neurons) whilst others arise in the many small nuclei present in the fillet path (Roller) and a few come from the anterior column of the spinal cord. During this course the mesial fillet gives collaterals to the reticular formation of the pons and later on to the midbrain (Flechsig, Bechterew, Held). At the same time the mesial fillet gradually assumes the shape of a flattened band, which in cross section has its long axis transverse instead of antero-posterior as in the interolivary layer. Further, the fibres coming from the nuclei of Goll and of Burdach are somewhat intermixed and have altered their relative positions, so that those of Goll predominate laterally (v. Gehuchten), or ventro-laterally in the fillet (Ferrier and Turner) (though v. Bechterew places them in the middle portion and v. Monakow and Tschermak chiefly in the mesial portion), whilst those of Burdach are most abundant in the remainder of the area. This flattened band in the pons, according to the excellent description of Obersteiner ([165] p. 408, *et seq.*), is made up of two kinds of fibres, one staining darkly by the Weigert-Pal method, the other faintly. The faintly-staining fibres appear first in the upper hypoglossal region, and lie ventro-mesial in the middle fillet; in the pons they quickly increase in number and lie mesially in the fillet, and as traced upwards in the pons they pass more laterally and leave the fillet for the stratum intermedium, whence they pass to the pes pedunculi. They are really descending motor fibres to the cranial nuclei (Hoche) which lie in the fillet area, and constitute the *Fusschleife* of Flechsig, the scattered accessory fillet of Bechterew, and the lateral pontine bundle of Schlesinger (who took them for central sensory fibres).

On the other hand, a few fibres lying most mesially in the fillet pass gradually at the upper border of the pons from within out (mesio-laterally) and from below up (caudo-frontally) circling around the surface of the crus to an unknown termination near the geniculate bodies and ventral thalamus. This has sometimes been called the accessory

fillet of Bechterew, but is perhaps better known as the *Bündel von der Schleife zum Fuss* or Spitzka's bundle, and being an ascending path is obviously not to be confounded with the *Fusschleife* mentioned above.

As the mesial fillet ascends in the midbrain it passes laterally and dorsally, lying at first ventro-mesial to the lateral fillet. As soon as the latter has ended in the posterior corpus quadrigeminum and the posterior brachium, the former passes still more dorsally, giving branches to the substantia nigra (Tschermak) and perhaps to the posterior corpus quadrigeminum (Tschermak [80], Wallenberg [81], Rothmann [83], Bruce, Sirleo [84], Vejas [79], but v. Gehuchten, p. 829, *contra*). The middle fillet certainly gives a number of fibres to the fillet layer of the anterior corpus quadrigeminum of the same side (Ferrier, Turner, Obersteiner, v. Gehuchten, Collier and Buzzard [156], Edinger [157], Schacherl and others), and possibly a few fibres to both anterior colliculi. Continuing frontally the remainder of the mesial fillet passes dorso-laterally to the red nucleus, presenting in section the shape roughly of a half-moon, and ends in the ventro-lateral part of the optic thalamus (see Fig. 12) of the same side (v. Monakow, Tschermak, v. Gehuchten, Wallenberg, Obersteiner, Bruce, Mott, Hatschek, Dejerine, Ferrier and Turner, Probst, Bechterew, Mingazzini, Schlesinger, Ceni, C. Mayer, Lewandowsky, Hatschek, &c.), and according to Cajal they terminate in extremely complicated arborisations around the cells of the lateral and central nuclei of the thalamus. According to Dejerine and Tschermak some also end in the internal medullary lamina and in the centre median. It is possible that a few collaterals or fine fibrils pass into the zona incerta and corpus subthalamicum (Obersteiner, Wallenberg). It may, however, now be accepted that no fibres pass through the posterior commissure to the opposite thalamus (Probst and Tschermak *contra*), nor into the peduncle of the corpora mammillaria (Wallenberg, Sirleo *contra*), nor into the lenticular nucleus of the corpus striatum (Flechsig, Darkschewitsch *contra*). It seems, moreover, quite certain from recent investigations that, contrary to some of the

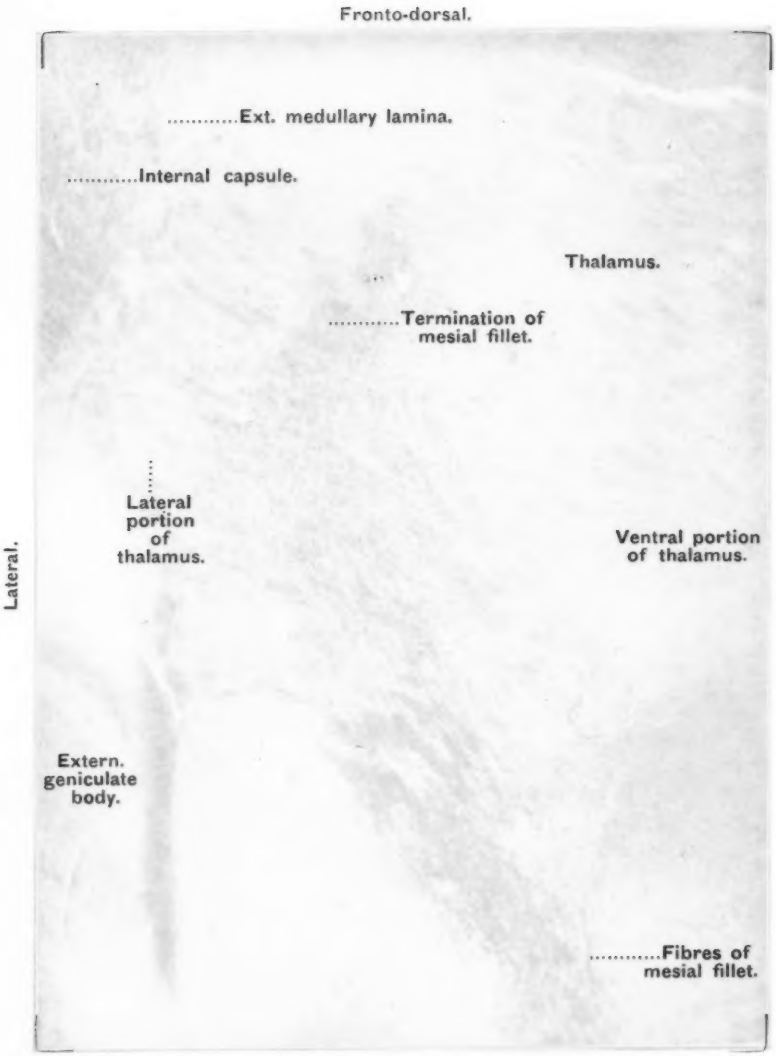


FIG. 12.

Photograph of a Marchi specimen.

Showing termination of the mesial fillet in the ventro-lateral (and central) portion of the optic thalamus in the monkey (*Macacus Rhesus*). (W.P.M.).

Fronto-dorsal.

Ext. medullary lamina.

Internal capsule.

Thalamus.

Termination of
mesial fiber.

Lateral
portion
of
thalamus

Ventral portion
of thalamus

Mesial.

Lateral.

Extern.
geniculate
body

Fibres of
mesial filler.

Caudo-ventral.

Fig. 1x

Photograph of a Marchi specimen

Showing termination of the mesial fillet in the ventro-lateral (and ventral) portion
of the optic thalamus in the monkey (Macacus Rhesus). (W. P. M.)

publications of Tschermak, Hösel, Sirleo and Flechsig, no fibres of the middle fillet pass directly on through the internal capsule to end in the cortex (Obersteiner, Marburg, v. Gehuchten, Probst, Lewandowsky).

To sum up, the mesial fillet or medullo-thalamic path arises in the nuclei of Goll and Burdach and totally decussates, to pass into the ventro-lateral and central portion of the optic thalamus of the opposite side. On its way it gives branches to the opposite inferior olive, to the reticular formation, to the substantia nigra, to the anterior corpus quadrigeminum, and possibly to the posterior corpus quadrigeminum, but it does not give fibres (crossed or uncrossed) to the cerebellum, to the nucleus lentiformis, to the homolateral thalamus, nor direct fibres to the cortex.

The Nature of the Afferent Impulses Passing up the Columns of the Spinal Cord.

The various afferent impulses having been conveyed to the spinal cord along the delicately differential peripheral mechanisms referred to above, the question arises how and along what paths are these impulses conveyed upwards in the spinal cord, and thence to the receptive centres in the brain, to render the organism conscious of changes in its environment. Needless to say, a copious literature has arisen on this subject, especially during the last half century, and it is only possible in this short communication to give a brief summary of the matter in its main essentials. In discussing this question, Rothmann refers briefly, firstly to the anatomical paths along which such impulses may pass, and then to physiological experiments in animals carried out by certain observers; and in his second publication, after excluding deductions drawn from widespread disease, gives important data obtained from results of accidental section of various tracts of the spinal cord in man.

Hemisection of the Spinal Cord.

The groundwork of this part of the question was laid by the great French scientist, Brown-Séquard [87, 88, 89], who

published in 1847, 1850, 1855, *et seq.*, the results of a series of masterly researches, and demonstrated that paths for touch, tickling, pain and temperature run, crossed or almost entirely crossed, on one side of the spinal cord, whilst muscular sensibility runs like the paths for voluntary movement and efferent vaso-motor impulses, uncrossed. He showed also that these facts were borne out by numerous examples in human pathology, and the Brown-Séquard symptom-complex became a more or less universally accepted fact in neuropathology. This view, however, was at first contradicted on many sides, and more especially by the great experimenter Schiff [90]. From numerous researches he concluded that all painful impressions were conveyed in the grey matter, and those of touch in the posterior columns. He stated that when all white matter in the thoracic region was divided, provided that a small bridge of grey matter was left, painful impulses from the hind limb were still appreciated. This view, though stoutly defended by Schiff, is now no longer held. And, indeed, in the course of his long investigations, Schiff himself modified very considerably his views of sensory conduction along the grey matter. Thus, after hemisection of the cord, he observed at first diminished movement on both sides, that on the operated side being persistent.[1] He also found homolateral hyperæsthesia, which rapidly diminished, and a sensibility lowered to pain and absent to touch. On the crossed (unoperated side), he found at first loss of all forms of sensation, then slight return to painful impulses and touch.

Weiss [92] found in hemisection, sensibility and movement almost completely lost on both sides. With rapid improvement in movement of the limbs on the opposite side to the lesion, sensation showed little if any improvement. Also Turner [101], after hemisection of the upper cervical region in monkeys, found in the opposite (heterolateral) arm, pain and temperature sense abolished but touch unaffected. The opposite leg was totally anæsthetic. The probable significance of this apparent contradiction is given below.

[1] To save space references to movement have been mostly omitted in the brief *résumés* above.

With time the anæsthesia diminished, but more so in the arm than in the leg, and Turner calls attention to the fact that monkeys with identical lesions vary in the amount of their sensory loss and in their power of restitution.

Mott [102] observed in the monkey diminished sensation on the opposite (unoperated) side, but even more affection of sensation on the side of hemisection, viz., absent touch, temperature, and muscular sensibility. Thus he found from a one-sided lesion affection of sensation on both sides, with touch and muscular sensibility passing chiefly on the homolateral side. Schäfer's [103] results also are roughly in agreement with the above. Similarly Botazzi [121] in dogs, in the operated side found anæsthesia to touch and temperature, and lessened appreciation of pain, but a still greater loss of painful sensation on the opposite side. Marshall [122] in the cat found tactile sensation running homolaterally. Langendorff [116] found after hemisection of the cord in the guinea-pig, diminished sensation on both sides, but a complete restitution after several weeks.

Further, the researches of Gotch and Horsley [104], of Osawa [98], and of Lewandowsky (p. 205), all tend to show that sensations are largely conveyed heterolaterally, but touch and muscular sensibility, and to a less extent pain, homolaterally.

Conduction in Posterior Columns.

As stated, Schiff held the view that tactile sensibility ascended in these columns. He demonstrated at a meeting of naturalists in 1858 that a rabbit with merely intact posterior columns (the rest of the cord transected) reacted quickly (sudden movement of head and ears, &c.), to the slightest touch on the hind foot or body, or by blowing on the skin, but remained quiet and unconcerned when the foot or tail were forcibly pressed or pinched. These results of Schiff were supported by the work of Herzen and Loewenthal [95], who found that division of the posterior columns blocked the impulses of touch, and also by the work of Bickel [96] and of Herzen [97], both of whom confirmed the above, and added that the impulses also of cold were no longer transmitted.

Osawa [166 and 98], however, showed in dogs, rabbits, and cats, that if the spinal cord be divided, leaving the posterior columns intact, no sensation nor voluntary movement were obtainable from below the level of section, and conversely he obtained the important positive results that if the posterior columns were divided, and the remaining structures left intact, the sensations of touch and pain persisted. Münzer and Wiener [99], as a result of experiment, considered that the posterior columns conveyed unconscious muscular sensibility; and Borchert [100], as a result of a careful experimental investigation, holds an intermediate view, viz., that division merely of the posterior columns produces no disturbance of general sensibility, that touch impulses are still conducted though blunted, and there is a diminution in the power of exact localisation of the spots stimulated. The writer's own experiments on division of the posterior columns, chiefly in the cervical region, in various monkeys (Macaque and Rhesus), so far as reliance can be placed on results obtained in the investigation of such equivocal points in lower animals, confirm Borchert's main conclusions. In addition, in the monkey a striking ataxy of the body and all four limbs was obtained from the moment of operation. This incoördination diminished with time, though was never entirely lost. As a *complete* division of the posterior columns was in no instance obtained without some degeneration being found later in the direct cerebellar tract, it was not possible to apportion the amount of ataxy caused by damage to the posterior columns. Further, the results of observation by a large number of careful clinicians of disease of the posterior columns in man (which are relatively larger and presumably of greater importance than in animals) must be carefully considered. Yet Rothmann (p. 77) for various reasons (widespread lesions, neighbour symptoms, concomitant disease, slowness of onset, compensation, &c.), lays stress on the fact that one cannot be sufficiently critical in their interpretation. Thus in tabes dorsalis parts of the posterior columns are often unaffected, whereas the posterior root fibres passing, to end in Clarke's column and other parts of the grey matter are frequently markably diseased, and even in

early tumours, pressure and local circulatory changes, &c., have to be taken into account. However, Leyden and Goldscheider [105] state that in sclerosis of the posterior columns in man, the sensation of touch remains, but is badly localised. In addition, Sherrington [106] states that "there is much in favour of H. E. Hering's view [107] that the posterior column serves to conduct centripetal impulses which, arising in the apparatus of movement, regulate by unconscious reactions the movements of the skeletal musculature," and most clinicians hold the view that disease of the posterior columns lowers the muscular sensibility, which is the cause of the characteristic disturbance of co-ordination (ataxy) in tabes, while most observers hold that this muscular sensibility (sense of passive position and of movement) passes homolaterally in the spinal cord.

Conduction in the Lateral Columns.

After Brown-Séquard's and Schiff's observations mentioned above, fresh knowledge was gained by work in Ludwig's laboratory, and more especially by Woroschiloff [91], who proved that the lateral columns were of dominating importance in sensory conduction and in voluntary movement. He made the important observation, that if in a rabbit the lower thoracic cord was transected, leaving the lateral columns intact, no disturbance of reflex activity between fore and hind limbs, nor of movement of the hind limb, occurred, whereas complete transection of the lateral columns removed both sensation and voluntary movement. Weiss [92] and Ott and Smith [93] confirmed and amplified these observations. But later Schiff, and also Osawa [96], proved that the lateral columns were not the only paths which convey sensory impulses. Also Bechterew and Holzinger [94] proved that division of both lateral columns in dogs prevented the conduction of painful impulses. Further, Ed. Bertholet [108], after a number of careful experiments, also demonstrated that the impulses of pain and temperature pass up in the lateral columns of the cord in the position occupied by Gowers' tract, and that the spino-cerebellar tracts do not play an indispensable rôle in the conduction of sensation.

On the physiological function of the spino-cerebellar (dorsal and ventral) tracts which run in the lateral columns, Marburg's results [109], after allowing for disturbance of the motor path, are in agreement with those carried out more recently, and with a stricter limitation of the lesion to these paths by Bing [110]. He states, however (p. 255), that some damage to the posterior roots was unavoidable, and so degeneration was observed "here and there" in the posterior columns. Bing shows that the fibres of the dorsal and ventral spino-cerebellar tracts which pass to the cerebellum, are of similar function (*op. cit.*, p. 253), viz., division of them in the second cervical segment produces hypotonicity and ataxy in the limbs of the same side, most marked in the central portions (hip and shoulder). The animal stands with the limbs of the operated side well abducted (hind limb more than fore limb), in walking tends to gyrate to the healthy side, and occasionally accidentally crosses (strongly adducts) its fore paws, sometimes, though less frequently, its hind paws. These symptoms, even with a bilateral lesion, rapidly improve and almost disappear in two or three weeks. During this period no disturbance of sensation was detected. Bing concludes from his researches (p. 268), that the spino-cerebellar tracts (cerebellar portion) subserve deep sensibility and convey unconscious impulses from the muscles, tendons, joints and bones to the cerebellum, and thus definitely affect reflexly (the intensity and duration of) impulses subserving tone and movement.

As regards cases in man which throw light on the sensory paths in the spinal cord, it would be impossible to attempt to summarise them in this paper. One must refer, however, to the careful collection and analysis of 137 such cases by Petrén [111], and the numerous references given by him in the literature of the subject. Rothman in his selection (*op. cit.*, p. 77) excludes all cases except those produced by accidental section (sword thrust, &c.) of portions of the spinal cord in man. Of these he has collected seventy, rejecting for his present purpose those cases without autopsy; of the ten remaining, five died too early to be of value, and he lays stress chiefly on three, which were care-

fully examined over a long period, and also microscopically later, viz., the cases of Jolly [123], Peugniez et Phillipe [124], and of Albanese [125]. The observation of Jolly's case showed that the sense of passive position was permanently abolished on the permanently paralysed side, whilst on the originally completely anæsthetic side, though painful sensation and the sense of passive position returned almost entirely, there was only a slight return of appreciation of pressure and absolutely no improvement in response to touch or temperature. The division of the cord at the eighth cervical segment, involved all tracts except the ventral portion of the lateral column, the lateral portion of the anterior column, and Burdach's column on the right side, and on the left side only a very small peripheral piece at the junction of Burdach and Goll's column escaped Hence the sensations eventually obtainable must have passed through the above unaffected areas. Notable points in Peugniez and Philippe's case were that, on the anæsthetic (left) side of the body sensations to pain and warmth returned to an appreciable extent though they were badly localised, yet the whole right half of the spinal cord, and the left half as far laterally as the lateral margin of the anterior horns, were divided. Hence the restitution of pain and warmth in the left leg must have occurred along paths which passed in the above restricted unaffected area in the left (homolateral) side of the spinal cord. In the case of Albanese, with complete division of the posterior columns, &c., appreciation of touch was perfect in the left leg, and absent in the right leg. In the cord the lesion divided all paths except those in the anterior column and anterior half of the lateral column of the right side and the mesial third of the anterior column of the left side.

Anterior Columns.

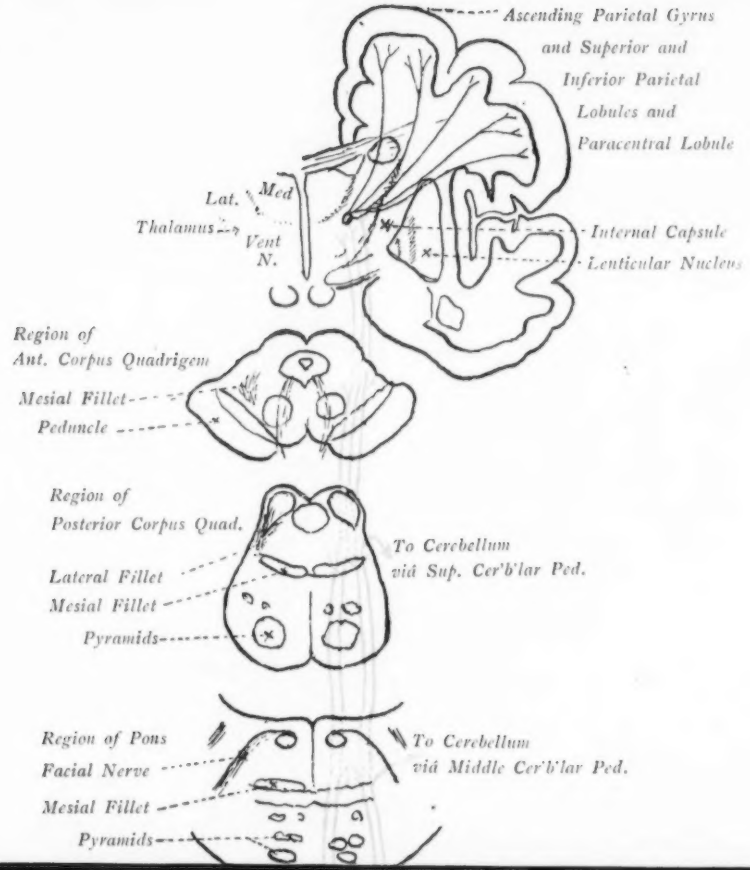
After division of the anterior and lateral columns Schiff showed that only pain impulses were transmitted, and Lewandowsky (p. 203), making the converse experiment, divided all but the anterior columns and found no sensory impulses transmitted at all. But Rothmann ([167] and [68], p. 49) found in dogs that section limited to the anterior

columns, produced quickly passing disturbances of tactility and considerable disturbances of equilibrium (ataxia) which were only partially compensated with time. He then made successive sections in the same animal of the anterior columns in the first cervical segment, and three weeks later of the posterior columns in the third cervical segment, and later of the lateral columns. Thus, in some instances, all centripetal paths were divided at one level or another, and yet in no instance, according to him, was sensation completely absent.

Conduction of the Different Forms of Sensation.

From the above, and similar observations, it follows that in the dog and lower animals no one form of afferent impulse is restricted to any one tract, nor even to one half of the spinal cord (see Fig. 13). For each more than one path seems open, and can with time be rendered available. The sense of touch seems most localised and passes in the homolateral posterior column and heterolateral anterior column; whilst, for instance, pain-impulses pass chiefly in the homolateral, though also in the crossed lateral column, and possibly also in the anterior columns; it appears that the different factors underlying muscular sensibility can pass in addition along the posterior columns (Rothmann, p. 76, Long [164].)

Here also we may remark that the methods adopted in the past for the investigation of sensation in animals, and even in man, have been open to serious drawbacks, and have not infrequently given erroneous results. The old method of testing touch, for instance, is a case in point. Further, in animals one can only judge of sensation produced by various stimuli, by the animal's response in voluntary and reflex movement; this again is influenced by concomitant changes in excitability or in motor paths. As an animal cannot answer "if" he feels a certain impulse, still less "how" he feels it, sensory investigation in animals must have its limits, though it has hitherto held the key to the solution of sensory conduction. Again, conduction solely along the grey matter, as advocated originally by


Ascending Parietal Gyrus
and Superior and
Inferior Parietal
Lobules and
Paracentral Lobule
Lat.
Med
Thalamus
Vent
N.
Internal Capsule
Lenticular Nucleus
Region of
Ant. Corpus Quadrigem
Mesial Fillet
Peduncle
Region of
Posterior Corpus Quad.
To Cerebellum
viá Sup. Cer'b'lar Ped.
Lateral Fillet
Mesial Fillet
Pyramids
Region of Pons
To Cerebellum
viá Middle Cer'b'lar Ped.
Facial Nerve
Mesial Fillet
Pyramids

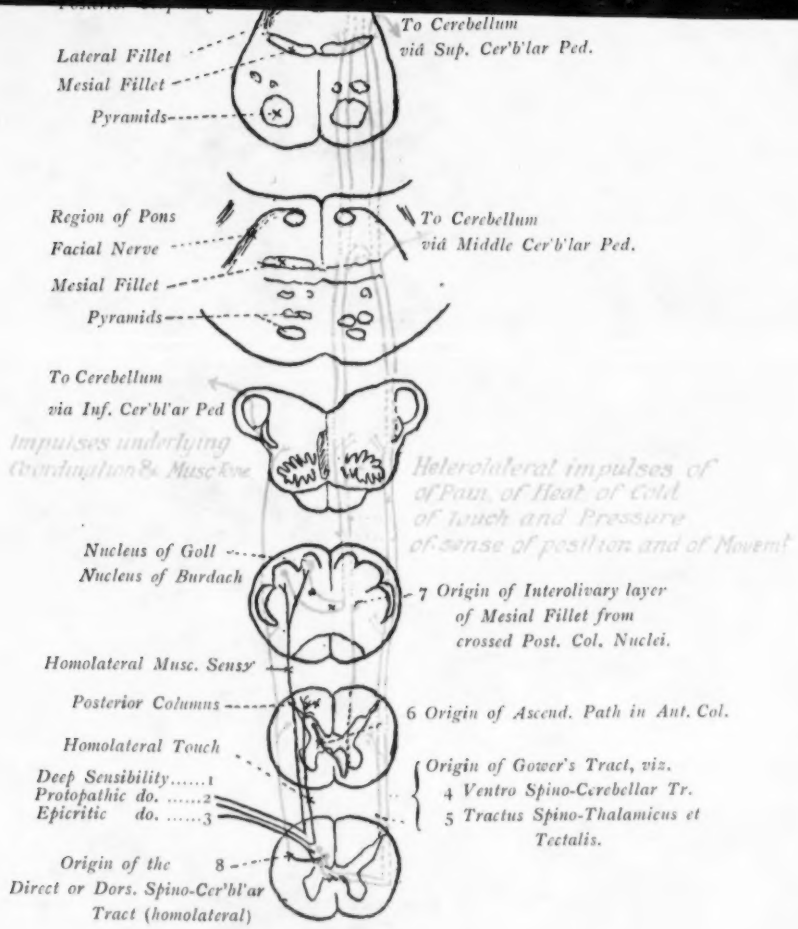


FIG. 13.—Diagram to illustrate the termination of the peripheral primary afferent neurones, the origin, course and termination of the secondary Central long paths (Spino-Thalamic) [to avoid complexity the short subsidiary paths and the associational cells (Bahn-Zellen) are not indicated] and the Tertiary Central Sensory Neurone (partly after v. Monakow). 1. Bundles of fibres subserving Deep Sensibility (pressure, pain from excessive pressure, Sense of passive position and of movement, locality). 2. Protropathic Sensibility (painful cutaneous, extremes of heat and of cold, diffuse response). 3. Epicritic Sensibility (light touch, discrimination of two points, fine grades of temperature, cutaneous localisation). Almost entirely Heterolateral:—4 and 5. Gower's Tract. (4) Ventral Spino-Cerebellar. (5) Spino-thalamic and Spino-tectal tract. 6. Origin of the ascending tract in the Anterior Column. 7. Origin of the Mesial Fillet. 8. Homolateral. Direct Cerebellar (Dorsal Spino-cerebellar tract of Flechsig). 9. Origin of the Tertiary Neurone in the Thalamus and passing to the Ascending Parietal, and Superior and Inferior Lobules of the Parietal Lobules.—*W. P. M.*

Schiff, does not take place in man or animals; for many cases of syringomyelia have shown (Spiller [170] and many others) that, with complete destruction of grey matter at various levels, conduction of all forms of sensation may exist. But it is obvious, as the anatomical arrangement of the posterior root fibres show, the paths of all forms of sensation must enter the grey matter of the spinal cord at one time or another, and it is clear that a block in the grey matter would interrupt an increasingly greater number of posterior root fibres, according as the lesion in the grey matter was of greater fronto-caudal extent.

This at once emphasises the fact that a lesion in the spinal cord has a local homolateral action, through interference with the crossing sensory impulses. A lesion involving the corresponding secondary afferent paths will produce heterolateral symptoms. This definite distinction between remote and local effects has frequently been overlooked, and led to innumerable mistakes in deduction and diagnosis. Further, as is well known, posterior root fibres ascend directly in the posterior columns from the most caudal parts of the cord to the posterior column nuclei in the medulla; other posterior root fibres terminate almost immediately on entering the cord, whilst fibres of intermediate length (as can be seen in almost any specimen of posterior root degeneration) are terminating at intermediate levels. Hence, again, the impulses of pain and temperature, which are conducted in fibres crossing early, soon enter a secondary path, and become heterolateral, whereas many impulses subserving muscular sensibility certainly run up through a large number of segments before entering their secondary path, and thus crossing to the opposite side. Whereas, I believe it is clear, from the cases quoted by Turner [101] and by others, where, after cervical hemisection, there was a heterolateral loss of touch in the leg, but a homolateral loss in the arm, that the fibres subserving touch do not cross so readily as those for pain and temperature. If so, their intermediate position would account for and largely explain the larger number of contradictory results (homolateral, heterolateral, and both) given

by various observers who have investigated the path of touch. In addition, in the following description we must bear in mind that there is no sharp line of demarcation between the anterior and the lateral columns, in fact, the distinction is quite ill defined, and this has sometimes led to error in description. Further, as regards future investigations, it cannot be too strongly emphasised, that not only our methods have been proved faulty by Head, but special experience and patience are required in those who venture to test sensation, particularly in its finer degrees. Neurology would be clearer if some of the above-quoted experiments had not been recorded, and, as has been said, it is easier to get truth out of error than out of confusion.

Further, Head has shown that each specific quality of sensation is conducted in its own restricted path or paths in the spinal cord (Marshall Hall address [112]). In other words, on embouching from the posterior roots into the spinal cord, a shunting and regrouping of the different specific qualities of sensation take place. Thus pain may be evoked peripherally by a needle applied to the skin (protopathic sensibility), or by excessive pressure applied to the muscles through a totally insensitive skin area (deep sensibility); yet in the spinal cord pain, if interrupted at all, is interrupted to all forms of sensibility. Similarly in the peripheral nervous system sensations, due to the application of extremes of heat or of cold are lost together, and, on regeneration, return together (protopathic sensibility), whilst the sense of fine discrimination of small grades of temperature, those usually called warm or cool, may be present or absent (epicritic sensibility), entirely independently of the other two. But in the spinal cord all degrees of heat are affected together and similarly all forms of cold. If the extreme of temperature be lost, the middle temperatures are also lost. Bearing the above in mind we may venture to sum up the conduction of the various forms of sensation in the spinal cord in man as follows :—

The Conduction of Painful Impulses in the Spinal Cord.

The posterior root fibres subserving this function after entering the posterior columns, rapidly end around cells in

the neighbouring grey matter. Thence a secondary path arises, most if not all the fibres of which cross almost immediately to the opposite side of the cord, and ascend in Gowers' tract. Here they lie mesio-ventral, and thus (see Fig. 10), especially when near their origin, lie partly in the lateral portion of the anterior column where it joins the lateral column, but chiefly, especially as they ascend, in the lateral column. Some of these special fibres certainly pass directly to end in the optic thalamus, whilst others terminate in the intermediate grey matter, and thus, by means of a series of short chains, afford secondary paths (Hilfsbahnen) to the same end station, which may supplement the direct path, or be made available after interruption of the direct path. Judging from the more or less perfect restoration of painful sensations after a complete hemisection of the cord, which has been abundantly proved in many cases in man and animals, the homolateral side of the cord must be able to supplement or replace the heterolateral path, and the impulses of painful sensibility here also pass up in the spino-thalamic portion of Gowers' tract (see Fig. 13). The inefficient and dilatory conduction of the latter is well illustrated by the symptom of allochiria (first mentioned by Boyer [113], also H. Munk [114], Obersteiner [115], Mott [102]), in which painful and thermal stimulation of a partially anæsthetic area is localised in the corresponding part of the opposite half of the body. The stimulus apparently uses the same path or paths for both sides of the body, and is erroneously referred from the cortex to the wrong side of the body. The symptom disappears as improvement continues. It has been shown in many cases that hemi-analgesia (or even hemi-anæsthesia) may persist for many years; in such cases it is invariably found (Rothmann, *loc. cit.*), that, at the onset, there was a more or less bilateral motor palsy, proving that both sides of the cord were to some extent involved. Painful impulses are not conveyed up solely in the grey matter, yet Schiff's original view is still held by some (Langendorff [116]) that each small bridge of grey matter is capable of conducting painful impulses, and doubtless here, as in all other instances, the

relation of intensity and duration of stimulus to the resistance of the path is of determining importance. In any case, however, each impulse of pain, like all afferent impulses, must pass through the grey matter somewhere, in a synapse, as explained above; but painful impulses, apart from the short transit which their posterior root fibres take to their end arborisations in the grey matter, do not ascend in the posterior columns. Lewandowsky ([61] p. 203) states that he has proved all long paths in the lateral column pass to the cerebellum, and that a direct path to the optic thalamus from the lateral column still remains to be proved. This is obviously not in agreement with the researches of many observers cited above. If impulses of pain and temperature pass up in Gowers' tract, as is universally admitted, impulses of these two sensibilities must pass to the cerebellum. Yet no disease restricted to the latter organ, or even absence of the cerebellum, has been proved to be associated with "any appreciable change in any form of sensation." "The sense of position of the limbs, stereognosis, and the correct appreciation of weight are also never impaired" (Stewart and Holmes [117]). Yet afferent, unconscious impulses of pain and temperature do pass to the cerebellum (see Kohnstamm [118]). The cerebellum also receives impulses underlying fatigue, as well as those impulses subserving tone and coördination (Marburg, Bing), which would certainly be utilised in the cerebellum, the central ganglion of the proprio-ceptive system (Sherrington [119]) for the regulation of muscle contraction and inhibition. It would seem then that unconscious afferent impulses of pain ascend to the cerebellum, and other impulses of pain ascend through direct long paths, and (at least, under certain circumstances) secondarily through subsidiary chain-like paths of short course to the optic thalamus, and thence, after passing a synapse, to the higher centres in the cortex.

Hence the conduction of painful impulses in the spinal cord of man occurs in health, chiefly in the lateral and slightly in the anterior column, and is almost entirely crossed, though, as shown above, the corresponding homolateral path may assume a more important rôle during the process of compensation in disease.

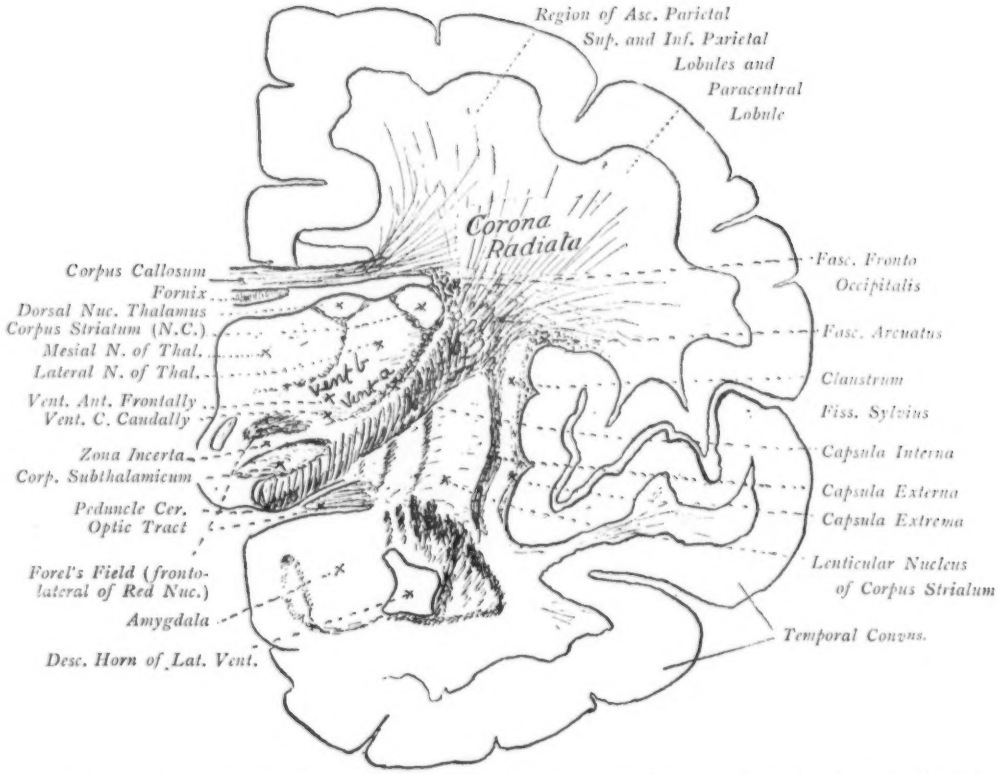


FIG. 14.—Diagram to show the general relations of the thalamic portion of the Sensory path and particularly the positions of the nuclei Vent Ant., Vent a, Vent. b, Vent. c, of Von Monakow.—Details in the text.—*W.P.M.*

Conduction of Thermal (Heat and Cold) Impulses.

These follow almost an identical course to that described for pain, but are slightly more, *i.e.*, almost absolutely, crossed. And usually, if one sense is affected, the other is also. Much of the discussion in the previous paragraphs about pain, applies to the present question (see Fig. 13). Petrén and Oppenheim have stated that these paths for pain and temperature lie so close together that they are always proportionately affected, but this statement cannot be accepted literally, especially in view of many cases recorded of disease in the cervical region. It must not be forgotten, too, that heat when intense may evoke pain. Neither is sensibility for cold always affected from the cord lesion when heat sensibility is affected (or *vice versâ*), though the dissociation is rare. It has already been stated that Herzen and Löwenthal [95] found in animals that one side of the body was anæsthetic to cold and not to heat, though they erroneously at that time thought the impulses were due to a block in the posterior columns. Similar cases have been recorded in man. For these and other reasons given above, it is clear that there are distinct and separate paths for the impulses of pain, of heat or of cold in the spinal cord (Head [112]), and that these different and specific qualities of sensation may be dissociated in an affection of the spinal cord; but as these paths are anatomically very closely associated from origin to termination, these three forms of sensation are usually affected, except at the confines of the anæsthetic area, to a like degree.

Hence the conduction of impulses of heat and of cold in the spinal cord occurs in man in separate paths, chiefly in the lateral column, and is almost entirely, if not entirely, crossed; though it would seem that the corresponding homolateral paths may, under certain circumstances, partly replace them.

Conduction in the Spinal Cord of Impulses for Touch and Pressure.

Rothmann states (p. 82) that conduction of impulses of pressure and of those for touch must be considered as being

quite apart. On the other hand, Petrén [111] always includes pressure and touch under one form of sensation (*Drucksinn*). The contradiction has been solved by Head [112], who shows that impulses of touch and of pressure may be conveyed in peripheral nerve mechanisms quite independently, whereas a lesion in the spinal cord which interferes with the sensation of touch will equally interfere with impulses which underlie sensibility to pressure, and that in the cord pressure is only an extreme form of touch. After division of the posterior columns, Schiff is quoted above as stating that the sensation of touch is abolished below the lesion, and Borchert that it is not abolished but is merely blunted and less well localised. This point of fine localisation is of importance, and yet has been often overlooked. Also cases of complete or nearly complete sclerosis of the posterior column in man have frequently shown that the sensation of touch is not abolished. In the latter cases the destructive process has, however, been of long standing, and time given for compensation and the opening up of accessory paths of conduction. It follows, from what has been mentioned above, that the accessory paths run in the heterolateral anterior column. The peripheral paths subserving the impulse of touch do not end in the spinal cord so readily as those of pain, of heat and of cold, nor do they extend through so many segments as the fibres which form the bulk of the posterior columns and convey some of the impulses underlying muscular sense. Hence at least as many as four or five segments are involved before the peripheral neurones subserving touch have fully ended, and including the intramedullary course, the corresponding secondary central neurones have fully passed the grey matter to the crossed anterior column. The number of segments thus involved would naturally vary with the level of the cord considered, being fewer in the thoracic region, and more in the lumbar, &c. Petrén holds that in addition to these paths the impulses of touch ascend in the lateral column, and several observers say in "all" the ascending columns and paths. But Rothmann (p. 82), with whom the writer is in entire agreement, says the impulses of touch pass definitely in

two paths, viz., the uncrossed posterior column and the crossed anterior column (see Fig. 13); division of both these paths abolishes sense of touch below, and when intact the two paths are sufficient to convey the sense of touch. Rothmann also adds that for the production of the sensation of touch, a certain amount of pressure sense is necessary (*gewisses Maas von Drucksinn*). Head has differentiated these in the periphery, and in the cord has shown they ascend together. But in view of the architectonic arrangements of the primary peripheral and secondary central paths as described above, it is of the greatest importance in the investigation of the sensation of touch to bear in mind the distinction between the "local" and the "remote" effects of a lesion in the spinal cord.

Hence one may conclude that in the spinal cord the impulses which underlie the sensation of touch ascend in the same paths as those for pressure, viz., in the uncrossed posterior column, and later in the crossed anterior column, and are present or abolished together.

Conduction of Muscular Sensibility in the Spinal Cord.

Muscular sensibility, which may "best be taken to include all reactions on sense arising in motor organs and their accessories," is "based on a specific set of sensations obtained by specific sense organs in muscles, tendons, joints, and all the accessory organs of movements" (Sherrington [106] p. 1002). Obviously it is made up of several components, in brief, those underlying the sense of passive position, of active and passive movement, of weight, of resistance, and possibly change in tension of the skin. As Petrén points out, further careful investigation of their component factors is much wanted. According to Rothmann ([68] p. 51), impulses underlying the muscular sensibility ascend in all three columns of the cord, and certainly least in the posterior columns. For he states, exclusion of the latter scarcely leads to any appreciable change in the muscular sensibility, whereas after division of the anterior columns it is considerable and not fully compensated. He also says, small portions of healthy

anterior lateral column sufficed in this connection in the dog (even with covered eyes) to enable him to maintain his equilibrium and to run securely (*sicher*). Most pathologists, however, hold the view that the posterior columns are the principal paths in the spinal cord for conduction of muscular sense. Afferent muscular impulses exert a great influence on muscular tonus, and there can be no question that reflex muscular movement is more easily elicited from stimulation of the posterior columns than from stimulation of any other columns or of the grey matter (Longet, Sherrington [106] p. 863, Page May [120].) In the control of reflex muscular tone and coördination afferent impulses passing in the spino-cerebellar tracts in the lateral column play an important *rôle* also; but as Bing [110] points out, these can be fully replaced along other channels in the dog in so short a time as two or three weeks. As cases of simple hemisection of the cord and many clinical cases show, the sense of movement and of passive position ascends homolaterally (Brown-Séquard). The posterior column fibres are homolateral as high as the medulla, and the dorsal spino-cerebellar tract is also homolateral. It is true that the ventro-spino-cerebellar tract of Gowers is in our opinion chiefly heterolateral, but Bing has already shown that in this connection its function largely coincides with that of the dorsal cerebellar tract, and that its loss of function is rapidly replaced along other paths.

To sum up, impulses underlying muscular sensibility are conveyed homolaterally in the spinal cord probably along two paths, unconsciously along the spino-cerebellar tracts of the anterior lateral column to the cerebellum,[1] and consciously, as well as unconsciously, along the posterior column of the same side. Thence they pass *via* the mesial fillet of the opposite side to the heterolateral thalamus and higher centres of consciousness in the opposite cerebral cortex.

[1] From the cerebellar cortex where the spinal fibres end fresh fibres arise, and a relay of fibres passes to the cerebellar nuclei and thence to the optic thalamus, &c., *via* the superior cerebellar peduncle, and *via* the direct sensory cerebellar tract of Edinger to the grey matter around Deiter's nucleus. These may possibly afford subsidiary paths for conscious passive position and movement in disease when the more direct path in the posterior column is blocked.

Conduction of Sensation in the Mesial Fillet.

As regards conduction in this path, numerous published cases have abundantly proved that when the mesial fillet is totally destroyed there always results complete anæsthesia to all forms of common sensation in the opposite half of the body, and according to the level of the lesion, fibres of the sensory cranial nerves are also involved on the corresponding side.

Thalamo-Cortical Path.[1]

The Thalamus.—As already shown, none of the fibres of the afferent path pass beyond the optic thalamus, and of those that reach so far frontally, all terminate in the ganglia of the ventral and lateral portions of the thalamus. It will be remembered that the thalamus forms the chief portion of the diencephalon, which is a direct product of the primitive fore-brain. Further, the various methods of investigation, especially those of secondary degeneration and of myelination, show that the thalamus is in abundant connection, by means of both efferent and afferent fibres, with practically every part of the cerebral cortex. Its minute anatomy is given in any of the well-known text books on the nervous system. A passing reference may here be made to the arrangement of the grey matter, which is broken up by layers of white fibres into more or less sharply defined masses called the ganglia or nuclei of the thalamus (see Fig. 14). The thalamus was formerly considered as made up of an anterior (or dorsal) nucleus, a mesial and a lateral nucleus. The internal medullary lamina separates the latter two nuclei and, forking above, serves largely also to limit the dorsal nucleus, whilst the external medullary lamina separates the latero-ventral nucleus from the internal capsule (posterior

[1] The remarks on the remaining portion (thalamo-cortical) of the afferent path must be brief. Only a few references can be given, but a full list of the literature bearing on the subject is to be found in Schäfer's Textbook of Physiology, or, more recently, by v. Monakow, in his excellent article "Ueber den gegenwärtigen Stand der Frage nach der Lokalisation im Grosshirn" in the *Ergebnisse der Physiologie*, also in the articles by Tschermak and Langendorff in the *Handbuch der Physiologie des Menschen* by Nagel, and also in the well-known works of Dejerine, Flechsig, Cajal.

limb). von Monakow [126] has since shown the advantage of dividing the lateral nucleus, as originally termed, into two main portions, which are here and there sharply defined, viz., the dorsal half or the lateral nucleus proper, and a ventral portion or ventral nucleus. Small lamellæ of white matter break up the latter into four well marked nuclei, which differ somewhat histologically and in their connections, viz., (1) the antero-ventral nucleus (vent. ant.) which consists of the most frontal portion of the ventral nucleus. Immediately caudal to this, the grey matter is split by irregular lamellæ of fibres into (2) a latero-ventral nucleus (vent. ant.), and (3) a meso-ventral nucleus (vent. b.; arcuate nucleus of Flechsig). The most caudal part of the ventral nucleus forms (4) the caudo-ventral nucleus (vent. c.).

Supposing the lateral nucleus divided horizontally into four equal segments, one finds the most frontal segment sends fibres to the three (upper, middle, and lower) frontal convolutions, the next segment to the upper part of the ascending frontal, the third segment to the upper portion of the ascending parietal convolution, and the most caudal segment to the superior and inferior parietal lobules. According to v. Monakow [126] the lateral nucleus is chiefly connected with the leg area, the ventral chiefly with the arm region. Further, though there is no actual sharp line of demarcation, he states that the antero-ventral nucleus (vent. ant.) is directly connected by fibres running in both directions with the second and third frontal convolutions, and partly perhaps also with the operculum and anterior portion of the ascending frontal convolution. The nucleus (vent. a.) seems similarly closely connected with the ascending frontal (arm region), and partly with the operculum. Whereas the caudo-ventral nucleus (vent. c.) and also the posterior portion of the mesoventral (vent. b.) are minutely connected with the supramarginal gyrus and the posterior portions of the ascending parietal convolution (arm area). It will be remembered that the mesial fillet ends largely in vent. c.

Hence fibres pass through the corona radiata (v. Monakow [151], Dejerine [152], Flechsig [153],) in both directions between practically all parts of the cerebral cortex and the

thalamus. Certain portions of the thalamus are intimately or almost exclusively connected with definite areas in the cortex as described above. The majority of these tertiary neurons of the central afferent path pass laterally into the posterior limit of the internal capsule, and then spread in a cone-like arrangement to the various parts of the cortex.

Certain functions of the optic thalamus.—Hughlings Jackson was the first to show so long ago as 1875, that a destructive lesion limited to the human optic thalamus produced complete hemianæsthesia on the opposite side of the body with impairment also of special sense and of motor power. Since then investigation, and more particularly experimental investigation, has produced an almost universal symptomatology as a result of destruction or stimulation of certain parts of the optic thalamus [128] in man and animals. The writer brought before the Physiological Society [129] results of experiments on the thalamus of sixteen monkeys; from these, as well as from the results of other observers, one fact stands out quite clearly, viz., the definite and constant association of hemianæsthesia of one side of the body, with a destructive lesion in certain parts of the opposite optic thalamus. The site of this lesion may be either in fibres of the second sensory neurons of the afferent path as they are terminating in the thalamus, or in the tertiary neurons of the afferent path which have arisen in the thalamus and are passing to the cortex, or in the intermediate grey matter. The other functions connected with this complicated subcortical ganglion are not within the scope of the present communication.

Localisation of General Sensation in the Cerebral Cortex.

The study of the localisation of sensation in the cerebral cortex, has proved much more complex and difficult than that of motion. The neopallium is not only extremely variable both in structure and function, but from small beginnings in the cyclostoma and selachians has been gradually evolved as the most complex portion of the whole nervous system. Physiologically, it is not a single organ, but a

collection of many, and each of different import. Its influence and control on the lower centres increases with the development of the organism in the animal scale; and whereas its removal in the lower animals leads to little change in muscular movement or sensation, in the primates its destruction leads to permanent loss of acquired voluntary movements, profound disturbance of sensation and of stereognosis.

Many authors have agreed that ablation of the frontoparietal region, particularly of the sigmoid area, in animals, leads invariably to sensory as well as motor disturbances of the opposite side of the body (more particularly Schiff, Munk, Goltz, Hitzig). Thus in a dog whose left sigmoid area has been removed, a touch or prick in the left foot is felt and localised at once, whereas similar application to the right foot is unfelt, and the dog makes no response until the stimuli are very much stronger or pain producing, when he makes every sign of uneasiness and discomfort, but completely fails to recognise the part which is being thus stimulated. Hence, after such a lesion, the sense of pressure and touch is seriously disturbed, and the power of correct localisation (topognosis) abolished. From numerous similar experiments to the above-named, and from the effect of excitation of the central (Rolandic) area in animals, it was supposed that the area in the cortex, excitation of which produced movement, was really sensori-motor (kinæsthetic, Bastian), and possessed not only the faculty of being excited from other parts of the cortex and thus causing voluntary and other movement, but that it was thrown into activity by impressions directly received from the ordinary afferent path, or, in other words, that it functioned as a region devoted to general sensibility. This view was for a time supported by a large number of distinguished neurologists, and H. Munk [144], amongst others, asserted that after extirpation of the arm and leg area in the Rolandic motor region of the cortex in the dog and monkey, the sense of touch and of pressure were permanently lost in the opposite limbs. He also stated that the power of correct localisation was especially easily lowered or abolished. (See also Mott [145]).

Schäfer [131], however, proved in the monkey that total removal of the area of the cortex, excitation of which produces movement in the hind leg, may be followed by no appreciable loss of sensation whatever, even when the ablation of the cortex is so complete that the corresponding limb does not participate in an epileptic convulsion of the remainder of the body. Thus Schäfer proved that paralysis of the limb resulting from a lesion of the cortex is not "necessarily" accompanied by any form of anæsthesia. From which it follows also that in such cases the paralysis of motion cannot be the result of loss of tactile or muscular sensibility, a point of importance in connection with some of the views held at that time, which supposed that paralysis was caused by the loss of muscular sense or loss of the consciousness called up by the imagining of movement. Therefore it is clear that if sensory localisation obtains, the corresponding motor and sensory cortical areas for each portion of the body are not identical. There may be more or less overlapping, but the confines of the correlated areas are not coterminous.

Researches bearing on this point were brought forward by v. Monakow [132], who showed that extirpation of the sigmoid region, including the lateral part of the gyrus coronarius in young dogs or cats, caused no degeneration or retrograde (Gudden) metamorphosis in the fillet portion of the afferent path. Neither has he ever found atrophy of the fillet in man as a result of a lesion restricted to the Rolandic area.

Sherrington and Grünbaum [133], working by the unipolar method of excitation in the highest apes, succeeded in showing, in a work which marks an epoch in cerebral localisation, that the true motor area, stimulation of which produces movement in the opposite side of the body, does not extend behind the fissure of Rolando. These researches, therefore, clearly showed that the post-central gyrus, whatever else its function may be, is not definitely part of the motor area; whereas it is well known from numberless experiments that ordinary sensation seems to have its maximum, if not its entire representation (Bechterew and

others), behind the central fissure of Rolando. Further, a fact of great significance was pointed out, viz., that though stimulation of the post-central gyrus (ascending parietal) does not directly evoke muscular contraction, it does increase the excitability of corresponding areas in the precentral gyrus (ascending frontal), and thus, as will be seen later, there would seem to be a close connection anatomically and functionally, between the post-central (presumably chiefly if not entirely sensory) area and the precentral motor area.

In agreement with this is the admirable and detailed work, more particularly of Bolton [134 and 135], Campbell [136], Mott [137], Watson [138], Brodmann [139] Flechsig [153] and others, with regard to the histological structure of various portions of the cerebral cortex in man and in the lower animals. Relying upon certain laminated and histological characters, these observers are able to mark out in the cerebral cortex areas which, from collateral clinico-pathological data, must be concerned especially with general afferent or senso-psychic or motor functions. These histological characteristics are of the greatest importance in their bearing on the localisation of cerebral function. Thus, to refer in one word to some of the results of Bolton and Watson, they show that the polymorphic layer of the cortex is (in the animal series) developed proportionally with, and would seem to subserve the lower voluntary functions of, the animal economy; the Betz cells and larger motor cells of the cortex are found to distinguish the motor areas, and readily serve to delimit these areas; whilst the granular layer of cells is especially developed in regions which all data would lead us to suppose are sensory. Not only that, but this layer is hypertrophied and even reduplicated in the visuo-sensory area, and serves sharply to delimit this from the visuo-psychic area (*cf.* also the audito-sensory and audito-psychic areas, the intermediate areas in the post-central region, &c.). The granular layer, therefore, by analogy subserves the reception or immediate transformation of afferent impressions, whether from sense organs or other parts of the cerebrum. Further, from histological investigation it is seen that the pyramidal layer

develops last, and in disease retrogresses first; its development and depth in the main are proportional to the mental power of the animal; it is proportionally atrophied with the amount of dementia or amentia, and it obviously forms the anatomical basis for the psychic or associational functions of the cerebrum.

Clinical results.—Schäfer ([130] p. 728), from his own observation in man, was in agreement with Charcot [140], that though paralysis of cortical origin is sometimes accompanied with disturbance of cutaneous and muscular sensation, the latter has no constant and necessary relation with lesions restricted to the cerebral motor area. In this connection see also Ferrier [141]. Further, the rare cases of purely sensory Jacksonian epilepsy would seem to prove that there must be a separate motor and sensory localisation in the cortex, though this view is opposed to the idea held by many observers on the evolution of the nervous system, and recently advocated by Horsley and C. K. Russell [142], according to which every part of the central projection apparatus must be sensori-motor. Yet as Mills [143] points out, that whilst it is true that sensation and motion are always represented in a complete reflex arc, spinal or cerebral, it does not follow that each of these functions is represented equally or in the same manner in each part of this arc. Numerous cases have been recorded by careful observers (Mills [143], Monakow (*op. cit.*, p. 628), Starr [146], Dana [148], Verger [147], Brodmann [139], Dejerine), which, after carefully sifting the facts, would tend to prove that the correlated sensory and motor areas are in distinct though adjacent portions of the cerebral surface. A lesion, for instance, in the middle of the ascending parietal gyrus caused diminution to all forms of sensation in the opposite arm and hand, which was more marked in the peripheral portion of the limb (Darkschewitsch [149]). Madden [150], with a lesion half an inch from the upper end of the same gyrus, showed that tactile sensation was much altered in the left hand and absent in the ring finger. The muscular sense was permanently absent and ataxia usually present. Further, Starr and McCosh [146],

in a case with a lesion at the junction of the posterior and inferior parietal convolutions distinctly posterior to the ascending parietal convolution, found loss of muscular sense in the opposite hand and forearm without any other sensory disturbance. From such and similar cases it would appear that the sensory area is situated in the post-central gyrus and neighbouring portions of the parietal cortex, and that lesions in this somewhat ill-defined area may produce disturbance of touch, of pressure, of localisation, of muscular sensibility (*i.e.*, the sense of passive position and of movement), and less readily perhaps of pain and temperature in the opposite side of the body, and that any of these qualities of sensation may be dissociated. Further, it would appear from cases quoted, particularly by Mills and Horsley, that the distal portion of the limb and the ulnar (post axial) margin of the limb are usually the earliest affected and the last to improve. Russell and Horsley further state that in affections of the sensori-motor area, *i.e.*, on both sides of the central fissure, the disturbances in such lesions as they describe are (1) of slight tactility, (2) of topognosis (the localisation was usually more proximal than the point stimulated); (3) of general orientation of the paretic part, and (4) occasionally of pain. From other cases it has been shown (Mills) that astereognosis may be absent in the part in which ataxia from the cortical lesion was persistent.

Stereognosis is a complex which requires numerous qualities of sensation for its production, and according to Mills and others it is a conception (stereognostic concept), and has a separate cortical localisation which is certainly postero-parietal in situation. Cases of cortical paresis without astereognosis, and cases of cerebral astereognosis without paresis, are now on record.

Further, the facts adduced by Mills would seem to prove that the cortical representation of cutaneous and muscular sensibility is independent of motor representation, that it surrounds (posteriorily and mesially) the motor zone, and that it is sub-divided into centres, each of which is anatomically or functionally correlated to a motor centre. Also, that stereognostic representation has its independent cortical

area, and is sub-divided after the manner of the motor and sensory areas.

Conclusions.

In the above short account of the sensory path merely the surface phenomena of the subject have been touched upon. The researches of Head, Rivers and Sherren, and of Head and Sherren, have not only placed old facts in a new light, but have enlightened a great deal of what was previously obscure. They are not only invaluable for the striking phenomena they so clearly reveal, but as an illustration of how to investigate, they are full of suggestion and help to others who may follow amongst the pitfalls of research. The brilliant histological researches of Cajal on the sensory nerve cells of the afferent path, &c., bristle with inviting questions which physiology cannot as yet answer or even scarcely consider. Whilst as regards the work of Rothmann, v. Monakow, Mills, and other distinguished neurologists mentioned above, they will be the first to suggest that our knowledge of the afferent path at present is little more than a series of hiatuses, and that minute imaginative and critical investigation is required at every link in the chain.

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W. PAGE MAY.

PROCEEDINGS OF THE NEUROLOGICAL SOCIETY OF THE UNITED KINGDOM.

A CLINICAL MEETING was held on Thursday, December 6, at 11, Chandos Street, Cavendish Square, at 8.30 p.m.

The following cases were shown:—

[1] Dr. JAMES COLLIER.—*A case of universal fixed and mobile spasm, with signs of pyramidal degeneration.*

A carpenter, aged 46 years. Family history unimportant. The spasm commenced in the left hand at the age of eighteen years, and spread gradually to the left foot, to the right foot, and last to the right hand. He was able to follow his trade until four years ago. During the last two years only has walking become increasingly difficult.

The psychic state is normal.

The fixed spasm is present in face, tongue, trunk and limbs.

The spontaneous involuntary movements are everywhere present, and are particularly marked in the toes. Some of the movements resemble myoclonic, choreic and athetotic movements.

Plantar reflexes extensor. Double foot clonus.

[2] Dr. T. D. SAVILL.—*A case of clonic spasm in a girl, aged 17 years, for diagnosis.*

The present condition of choreiform movements affecting the diaphragm, neck, and upper extremities, has existed for about seven months, in spite of all treatment. There is a history of attacks of the same kind of choreiform movements since the age of 7, when they were said to have been started by seeing another girl with St. Vitus's dance. The movements continued on and off, with intervals of a month, up to the age of 12. There was no history of hysterical fits or paralysis. She was admitted to the

National Hospital at the age of 12, with jerking movements of the head, face, and hands. The general health seemed good. She had a barking cough. The reflexes were normal. The treatment at that time had consisted of isolation, rest, and milk diet. The movements disappeared, but, after she had been allowed to sit up and read a book, on one occasion they returned.

The constant recurrence and final persistence of the movements suggested an organic lesion of some kind.

[3] Dr. E. Farquhar Buzzard.—*A case for diagnosis.*

J. L., male, aged 58 years. Builder's labourer, who has not worked with lead for some years. Thirty-five years ago venereal disease, but no sore. Eleven years ago a blow on right hand, without evident injury; a week later, wasting and weakness of right hand and wrist, which has been disabled ever since. Four years ago a similar condition began in the left hand, but recovered rapidly. During the last six months atrophic paresis has again involved the left forearm and hand. For four or five years has used a catheter night and morning; previous to that he had suffered for some time from retention and incontinence. For some years has had lightning pains in the bones and muscles of the legs.

Present condition.—Pale and thin. Right pupil larger than left. Neither reacts to light, and there is very little reaction on accommodation. Complete atrophic palsy of the intrinsic hand muscles, the flexors of the wrist and the extensors of the fingers on the right side. A similar condition on the left side, but much less marked. Legs strong. Arm and leg tendon jerks brisk. Plantar responses flexor in type. Some slight analgesia and defective thermal sensation over the legs. Tactile sensation good. Some dorsal kyphosis; trophic changes in right hand and nails. Slight unsteadiness with eyes shut and feet together. Discs healthy.

[4] Dr. Travers Jones (for Dr. Wilfred Harris).—*Case of juvenile tabes showing a "Charcot's joint."*

Ada T., aged 22 years, single. Classical signs and symptoms of tabes present, viz.: Argyll-Robertson pupil, lightning and girdle pains, loss of knee jerks, and Romberg's symptom; no signs of congenital specific disease; signs of past rickets. Knee is a typical Charcot's joint; doubtful history of interstitial keratitis in brother.

[5] Dr. Frederick E. Batten.—*A case of spinal new growth.*

Lavinia M., aged 14.

Family history.—Negative.

Previous history.—Healthy baby. Measles and mumps, aged 8 years, which left her with a lump in right side of neck, which was operated upon at St. Bartholomew's three and a half years ago. Nature of gland—lymphadenomatous (?).

Present history.—Three years occasional pain in back of neck on suddenly moving or jumping about. No radiation of pain.

Eleven months ago—stiffness of right calf muscles, weakness of right knee; difficulty in abducting right leg to mount bicycle.

Ten months ago—weakness of fingers of right hand. No paresthesia in hands.

Since then the weakness in right leg has grown progressively more, until three months ago the left leg further became weak; both legs often tremble violently when extended, less when bent.

No symptoms as regards eyes or sphincters.

Constitution.—Healthy-looking child. Operation scar right neck, beneath which are a few glands, firm, discrete, non-fluctuating, movable.

Cardio-vascular, respiratory, and abdominal systems normal.

Special senses.—Normal.

Cranial nerves.—Pupils equal, react normally, dilating to shade. Remainder normal.

Motor.—General musculature slight, tone fair, *slight wasting* of thenar and hypothenar eminences and interosseal muscles, especially first dorsal on both hands.

Marked trepidation of right lower limb, occasionally of the left, on manipulation. Slight drop-foot, right side.

Movements.—Weakness of flexors of wrist, right and left, with radial deviation of hand.

Movements of thumbs right and left very limited. Hip and knee movements all present, but reduced. Dorsiflexion of right ankle absent.

Gait.—Spastic, right more than left.

Sensory.—Relative loss to both tactile and painful impressions on limbs and trunk below and including cervical segments seven and eight.

To heat stimuli, a patch on left trunk from third costal cartilage to one inch below umbilicus, where impressions are not received.

Position sense.—Normal.

Reflexes.—Upper extremity all present, right and left. Abdominal—absent, right and left.

Lower extremities.—Knee-jerks, ankle-jerks, with patellar and ankle clonus present, right and left. Plantar—extensor right and left. Sphincters *nil.*

Electrical.—Loss to faradism in small hand muscles right and left. Incomplete polar change, K.C.C. = A.C.C. to galvanism.

Radiogram.—Some blurring and indefinition of right side of vertebræ, fifth to seventh cervical, and of corresponding intervertebral discs.

Spine.—*Nil*, externally.

[6] Dr. FREDERICK S. PALMER.—*Double primary optic atrophy* (*complete*).

(*a*). H. J., aged 33, single. Is a pork butcher, but has not done any work since April last. He has been blind with the right eye since August, 1905, and with the left since May, 1906. The loss of sight came on gradually in both eyes, without headache or vomiting. He is now completely amaurotic — unable to distinguish night from day. He contracted specific disease fourteen years ago, for which he was treated by a chemist for two months, and never had any medical advice. Both discs are atrophic, and the vessels fairly large. His gait, when led, is normal, and he stands well alone. There is no marked inco-ordination of the upper limbs; the knee-jerks and ankle-jerks are present, but diminished. There is some degree of ptosis, and his eyeballs appear to be rather prominent, but the ocular movements are perfect. Last May he was an in-patient for a fortnight in one of the ophthalmic hospitals, and lumbar puncture was performed. About two drachms of clear cerebro-spinal fluid was withdrawn. No evidence of lymphocytosis was discovered on examination. He has never had any visceral crisis, but in July and August last he complained of severe lightning pains in both legs and in the left arm. Since then he has been quite free from any recurrence of the attacks. He has never had any sphincter troubles, nor is there any alteration in sensibility to rough testing.

(*b*) *Primary progressive myopathy* (*?*)

H. S. has been under observation for a few weeks. He is 33 years of age, single, a teacher in one of the L.C.C. schools, and continued his work up to the end of last August. No history of familial nervous disease in his ancestors, or collaterals. An

uncle, by marriage, is said to have developed tabes at the age of 60. He had measles and whooping cough in childhood. No other illness until nine years ago, when he had a bad attack of sciatica, which obliged him to remain in bed for a fortnight. Since then, the pain has recurred at varying intervals, but lasted a much shorter time, not more than one or two days on each occasion. Five years ago (in 1901) he began to complain of stiffness, with rheumatoid pains in the muscles of the neck and shoulders, which have never entirely disappeared. Atrophy of the muscles was first noticed in 1903, and has been steadily progressive. His face is thin, and suggestive of myopathy, but there is no weakness of the orbiculares. He closes both eyelids well, and can whistle. There is rigidity, and very considerable wasting of the muscles of the neck, which looks lengthened. Marked atrophy of the muscles of the shoulder girdles, proximal segments of both upper limbs, and of the pelvic girdles. His shoulders droop, and there is some winging of the scapulæ. The muscles of the distal segments of the upper limbs and those of the hands are well preserved, including the supinators. His gait is normal, there is no evident lordosis, and his knee-jerks are present. Both feet show talipes valgus. The electrical reactions are diminished to both currents, but the R.D. has not been obtained.

[7] Mr. Donald Armour.—*Endothelioma of the left frontal region: removal by operation: recovery.*

A. C., male, aged thirty years, admitted to the National Hospital under the care of Sir William Gowers.

Family history, unimportant.

Previous history.—Bicycle accident, July, 1905. Unconscious for a few minutes, then dazed for hours. No fracture of cranial bones. October, 1905, began to suffer from attacks of transient amblyopia, lasting a moment at a time. At first only one or two a day occurred, latterly a dozen or more. Vision has been failing for reading. No diplopia. The attacks of blindness increasing in duration. For six months more or less constant headache, frontal and bilaterally equal. Has never had any sickness or vomiting. Considerable loss of memory during the last three months, both for recent and remote events. Some loss of orientation. No change in temper or affections. Constant drowsiness for the last two months.

On admission, well-developed, healthy-looking man. Sleepy, but easily roused, and always clear in his cerebration. Frequent

attacks of blindness. Considerable frontal tenderness on percussion, more marked on left side. No fits, no aphasia, no confusion in words. Smell defective on both sides, greater on left. Visual acuity: right, $\frac{6}{20}$ths; left, $\frac{6}{6}$ths. Optic discs: well-marked double optic neuritis, with hæmorrhages, wrinkling of retina, changes at macula. Swelling: left + 6 D., right + 5 D. No alteration in visual fields to rough test. No ophthalmoplegia. No facial asymmetry or weakness. No difference in range or power of voluntary movement. Fine tremor in left outstretched arm. No inco-ordination; no astereognosis. Topognosis is accurate right and left. No objective alteration in sensation. Double plantar flexion, no ankle clonus; knee-jerks present and equal. No sphincter trouble; gait normal; no ataxia; no vaso-motor or trophic changes.

[8] Dr. Wilfred Harris.—*Traumatic Sprengel shoulder.*

Palsy of mid and lower trapezius, levator anguli scapulæ and rhomboids on left side, due to muscular strain.

[9] Dr. Leonard Guthrie.—*Poliomyelitis simulating Erb's paralysis from injury to fifth and sixth cervical nerves.*

Boy, aged $3\frac{1}{2}$ years, has palsy and atrophy of right deltoid, biceps, brachialis anticus and supinator longus. Supra- and infra-spinati are less affected.

The sternal half of pectoralis major, trapezii, scaleni levator anguli scapulæ, rhomboids, teres major and latissimus dorsi escape. The triceps is wasted, but not paralysed.

In the forearm all muscles are intact except supinator longus. In the hand the first dorsal interosseous is wasted.

At 13 months of age a small superficial abscess over the right mastoid was opened. No anæsthetic was used. Next night both arms were noticed to twitch, and on the following morning the right upper extremity was found paralysed as at present.

No history of traumatism.

[10] Dr. S. A. K. Wilson.—*A case of torticollis in a child.*

(*a*) L. R., aged 7 years, was admitted to the National Hospital, under Sir William Gowers, on November 9, 1906.

Instrumental birth, and on third day had convulsions. Recovered without apparent sequelæ. Mother says there was always slight weakness on right side. Always tendency to let things fall from right hand. Never been able to form letters

properly with right hand, when learning to write. Nothing noted in right leg. Not backward at school.

Six months ago, was struck on right side of neck by a strap and buckle, and complained of local pain. During next few weeks, complained of a stiff neck, and began to hold his head tilted a little to one side. Two months ago, involuntary movements of head to left began, and soon after it was noticed that the right arm was moving spasmodically. About a week ago, mother noticed synchronous elevation of eyebrows.

The movements are well seen as patient walks, the head being flexed and rotated in a tonic manner to left. Occasionally the right shoulder is raised. The right arm is frequently adducted and inverted. When the spasm is marked, the left platysma takes a share in the movements.

At present, no definite evidence of old organic disease right, as suggested by the history.

(*b*) *A case of unilateral tremor.*

A. N., aged 49 years, was admitted to the National Hospital under Dr. Collier, on September 24, 1906.

Nil ad rem in previous history.

November, 1905, had a "fainting attack," without actual loss of consciousness, and found thereafter that her legs seemed weak. At same time, began to complain of a smarting, tingling pain in right thigh. These have not altered.

June, 1906, without any premonition whatever, noticed one day that left arm, and, to a less extent, left leg, began to shake, and there has been no change in this tremor either.

Patient is rather emotional, with tendency to *rire spasmodique*. Speech inclined to be a little slurring. Optic discs are normal. Well-marked nystagmus on lateral deviation to left and right, rather coarser to left, the movement of the eyeball is a rolling one, not in the horizontal plane, but rather oblique.

On volitional movement, left side of face moves less well than right; on emotional movement, the converse is observed. Tongue protruded with curve convex to right, and it is tilted on to its right border.

Constant rhythmical oscillating tremor of left arm and left leg, seen best when limbs are unsupported; no rigidity in these limbs.

Paræsthesia in right leg, with slight diminution of sensation in corresponding areas. Deep reflexes are brisker on right side.

Double flexor response.

Gait feeble.

[11] Dr. G. NEWTON PITT.—*A case with violent ataxic movements, of cerebellar type, of uncertain origin.*

(*a*) Man, aged 30 years, who fell down fifteen feet into a barge eight years ago, but was able to continue his work. This was followed by diplopia, and two days later by weakness of the legs, and shortly after movements of his hands. This gradually cleared up at the London hospital.

Six years ago the difficulty in walking and the movements in his hands became worse, but again improved under treatment.

He worked till sixteen months ago, but has gradually become worse.

His gait is impossible unless he is supported, and then it is slow, ataxic, and slightly spastic.

He generally sits in an arm-chair grasping the arms when his hands and head are steady. Directly he attempts to move his hands has violent movements, chiefly of flexion and extension of his forearm, and he is quite unable to touch his nose or to feed himself unless he grasps one hand with the other when he steadies it. Yet he can hold his arm out steadily and vigorously if he has even slight assistance afforded him. There is nystagmus laterally. Muscular power in arms good, they are generally vigorously contracted. Knee-jerks brisk, extensor plantar reflexes. Sensation normal, except for slight numbness in the fingers.

He has had attacks of vomiting, lasting for some days, and very occasionally headaches. Speech probably unaltered.

When he attempts to drink, his head oscillated up and down, unless it is supported. There is a large oval patch of choroidal atrophy with pigment round the margin near to the disc in the left eye, of a toxic or possibly traumatic origin.

(*b*) Man, aged 45 years. Three years age he noticed that his left leg began to give way suddenly when he was walking. This occurred more frequently, and now it always gives way at the knee. He had a large bursa removed from the popliteal space without improvement. When lying on a bed all the muscles appear to be efficient; his knee jerks and plantar reflexes are normal. He has no pain or defect of sensation.

The cause is obscure.

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Rules

AND

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OF THE

Neurological Society of the United Kingdom

1906

WITH THE

Annual Report of Council, and Balance Sheet for the Year 1905

London
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Neurological Society of the United Kingdom.

REPORT OF THE COUNCIL FOR 1905.

FEBRUARY 1st, 1906.

The Council has much pleasure in reporting the continued prosperity of the Society, which now numbers 253 members.

The Council regrets the loss of one of its original members, Sir John Burdon Sanderson, Bart., who died on November 23, 1905.

The Presidential Address, given on February 16 by Sir John Batty Tuke on "The Relations of the Lunacy Laws to the Treatment of Insanity," was fully reported in the Spring Number of "Brain."

The abstract of the Proceedings shows the scope and character of the work of the Society during the past year.

The Summer Meeting was held on Saturday, June 24, at the National Hospital, and the metropolitan members gladly embraced the opportunity of entertaining the extra-metropolitan members who were able to be present. The meeting was well attended and was successful both as a scientific and as a social gathering.

The question of amalgamation of the Society with the Union of Medical Societies has been carefully considered by the Council, and, at a Special Meeting of the Society called for the purpose on November 18, the Council recommended: —"That the Neurological Society of the United Kingdom join in the scheme for amalgamation of Medical Societies."

The financial state of the Society has improved during the past year under the careful treasurership of Dr. James Taylor.

The Council has much satisfaction in announcing that Sir Victor Horsley has consented to deliver the Hughlings Jackson Lecture during 1906.

Proceedings of the Society during the Year 1905.

February 16.—Sir JOHN BATTY TUKE, Inaugural Address, "The relation of the lunacy laws to the treatment of insanity."

March 30.—Clinical Meeting. Dr. ORMEROD (1) "Case resembling Friedreich's Disease, but exhibiting extensive anæsthesia." (2) "Case for diagnosis (?) progressive disease of pons and peduncles." Dr. GORDON HOLMES, "Case of mid-brain lesion with unilateral tremor." Dr. WILFRID HARRIS, "A case of chronic chorea." Dr. A. F. VOELCKER, "Encephalitis cerebelli." Dr. T. D. SAVILL, "Two cases of generalised tonic muscular spasm allied to paralysis agitans in middle-aged men." Dr. LEONARD GUTHRIE, "Primary atrophy of optic nerves, with partial ophthalmoplegia and cerebellar gait." Dr. HARRY CAMPBELL, "Two cases of tremor." Dr. HENRY HEAD, "Case of Huntingdon's Chorea."

May 18.—Ordinary Meeting. Papers read by: Dr. E. F. TREVELYAN, "A note on tabes dorsalis occurring in husband, wife, and daughter." Drs. C. R. BOX and E. FARQUHAR BUZZARD, "An unusual case of combined degeneration of the cord, associated with anæmia." Dr. T. GRAINGER STEWART, "Some points in the diagnosis of tumours of the frontal lobe."

June 24.—A Special Meeting was held at the National Hospital, Queen's Square. On this occasion Extra-Metropolitan were entertained by Metropolitan Members of the Society.

In the morning, Clinical Cases were shown by (1) Dr. C. E. BEEVOR (*a*), "Progressive muscular atrophy—Immobile pupils," (*b*) "Peroneal type of family muscular atrophy," (*c*) "Mesencephalic lesion." (2) Dr. J. H. BRYANT, "Two cases of peroneal type of family muscular atrophy." (3) Dr. GEORGE OGILVIE, "A case of tabetic gait beginning at the age of 64." (4) Dr. CAMPBELL THOMSON, "A case of 'tremor.'" (5) Dr. T. SAVILL, "A case of ataxy in a woman, aged 40, of twenty years' duration." (6) Dr. D. B. LEES and Mr. BALLANCE, "Case of hydrocephalus after post-basic meningitis —operation—recovery." (7) Dr. THOMAS BUZZARD, (*a*) "Case of mesencephalic lesion," (*b*) "Case of muscular wasting with unusual symptoms." (8) Sir VICTOR HORSLEY, "Three cases after removal of cerebellar tumours." (9) Dr. E. F. BUZZARD, "Case for diagnosis." (10) Dr. F. E. BATTEN (*a*) "Congenital affection of sixth, seventh, and twelfth cranial nuclei," (*b*) "Case of tremor of right arm associated with epilepsy," (*c*) "Case of acute ataxia." (11) Dr. A. F. VOELCKER, "Two cases of ataxia in children." (12) Dr. LEWIS JONES, "Case of injury to the brachial plexus." (13) Dr. HENRY HEAD, (*a*) "Case of myoclonus," (*b*) "Huntingdon's chorea." (14) Dr. RISIEN RUSSELL, "Unusual case of disseminated sclerosis." (15) Dr. HARRY CAMPBELL, "Two cases of cerebral tumour." (17) Dr. LEONARD GUTHRIE, "Case of acute ataxia."

In the afternoon, Communications were made by Dr. ALEXANDER BRUCE, "On the distribution of the cells in the intermedio-lateral tract in the dorso-lumbar region of the spinal cord." Dr. MOTT, "On the brain and nervous system in trypanosome infections, including sleeping sickness, and the part played by diplo-streptococci." Sir VICTOR HORSLEY, "Cerebellar lesions."

Specimens were exhibited by—Dr. C. E. BEEVOR, Injected brains showing the vascular supply of the anterior, middle, posterior cerebral, and of the anterior choroid and posterior communicating arteries. Dr. L. HUMPHREY, The parathyroid glands in Graves' disease. Dr. W. B. RANSOM, Syringomyelia with neuro-epithelioma of the spinal cord. Dr. STANLEY BARNES (i.) Healed tubercular meningitis; death from secondary hydrocephalus. (ii.) Sarcomatosis of spinal pia, secondary to cerebral tumour. Dr. R. T. WILLIAMSON (i.) Degeneration in the intramedullary fibres of spinal posterior nerve roots in tabes dorsalis, diabetes mellitus, and intracranial tumours; (ii.) Changes in the spinal cord associated with severe anæmia. Dr. E. FARQUHAR BUZZARD, The morbid anatomy of myasthenia gravis. Dr. HALE WHITE and Dr. F. A. BAINBRIDGE, Specimens from case of caisson disease. Dr. LEWIS JONES (i.) Twelve photographs of tracings of current waves of medical coils, to illustrate the variety of curves of those currents; (ii.) Induction coils to illustrate the curves of current waves; (iii.) Mechanical interrupters for the production of interrupted currents for diagnostic purposes.

Various other specimens from the Museum of the National Hospital were shown.

October 26.—Clinical Meeting. Dr. H. G. TURNEY, "A case of generalised spasm, most marked in the muscles of the back." Dr. ERNEST JONES, "Bilateral contractions simulating pseudo-hypertrophic paralysis." Dr. COLIN K. RUSSELL, "Case of juvenile type of general paralysis of the insane." Dr. F. E. BATTEN, (*a*) "Ulnar neuritis with fusiform enlargement of the nerve, occurring fifteen years after injury to the elbow"; (*b*) "Defect in upward and downward movement of the eyes. Mental impairment." Dr. FARQUHAR BUZZARD, "A boy with some cranial nerve palsies." Dr. LEONARD GUTHRIE, "Two cases of (?) early Friedreich's disease."

November 18.—A Special Meeting was held at which it was resolved "That the Neurological Society join in the amalgamation of Medical Societies, provided that there be a separate Neurological Section in the New Society."

November 23.—Ordinary Meeting. Paper by Dr. F. J. POYNTON and ALEXANDER PAINE, "Some investigations on acute rheumatic nervous affections."

December 7.—Pathological Meeting. Paper by Dr. JOHN TURNER, on "The pathological anatomy of idiopathic epilepsy."

Mr. J. HERBERT PARSONS, "Brain from a case of orbital meningo-encephalocele."

Dr. GORDON HOLMES, "Specimens from a case of pseudo-hypertrophic paralysis.

Dr. F. E. BATTEN, "Two cases of acute anterior poliomyelitis."

Dr. LEONARD GUTHRIE, "Congenital hydrocephalus and cerebellar tumour (card specimen)."

BALANCE SHEET. 1905.

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				Less Contributions for Illustrations from Authors of Papers 14 1 0			
					46	15	2
				Contributions for Illustrations to "Brain"	20	5	6
				Medical Society of London, Rent	15	15	0
				Mr. Bethell for Refreshments, &c.	7	0	0
				Petty Cash	3	7	6
				Balance	89	13	0
	£334	17			£334	17	11

Audited and found correct, January 18, 1906 { E. FARQUHAR BUZZARD. J. HERBERT PARSONS.

JAMES TAYLOR, *Hon. Treasurer.*

RULES.

1.—The Society shall be called the NEUROLOGICAL SOCIETY OF THE UNITED KINGDOM.

2.—The objects of the Society shall be to promote the advance of Neurology and to facilitate intercourse amongst those who cultivate it, whether from a Psychological, Physiological, Anatomical, or Pathological point of view.

3.—The Society shall consist of Ordinary, Corresponding, and Honorary Members.

4.—Any one engaged in researches bearing on Neurology, or manifesting interest in such researches, shall be eligible for the Ordinary Membership.

5.—Men of distinction in Science, who have contributed to the advancement of Neurology, shall be eligible for the Honorary, or for the Corresponding Membership.

6.—The number of Honorary Members shall be limited to six, and that of Corresponding Members to twelve.

7.—Honorary and Corresponding Members shall have the right of attending the Meetings of the Society and of taking part in them.

8.—Honorary and Corresponding Members shall be elected by the Society on the recommendation of the Council.

9.—Candidates for Ordinary Membership shall be recommended by at least three Members of the Society, who shall append their names to a printed form supplied by the Secretaries to any person seeking Membership.

10.—The Council shall nominate candidates for election into the Society as Ordinary Members from among persons so recommended.

11.—The names of Candidates nominated by the Council shall be submitted to the next Meeting of the Society, and shall be balloted for at the next subsequent meeting, one black ball in ten excluding.

12.—The Annual Subscription of an Ordinary Member shall be One Guinea, due in advance.

13.—The Treasurer shall send to each Member, shortly before the Annual General Meeting, a notice reminding him that his Subscription becomes due on that day.

14.—Non-payment of the Subscription within twelve months after it is due shall be considered as equivalent to resignation.

15.—The Council shall consist of a President, two Vice-Presidents, two Secretaries, one Treasurer, the Editor of "Brain," and twelve Councillors, of whom not less than three shall be extra-Metropolitan Members.

16.—Five Members of the Council shall form a quorum.

17.—The office of President shall be tenable for one year, and be entered upon at the beginning of each year by the Senior Vice-President.

18.—The office of Vice-President shall be tenable for two years, one Vice-President being elected every year.

19.—The Secretaries and Treasurer shall be elected annually with eligibility for re-election.

20.—The Councillors shall be elected for one year, and not more than eight shall be eligible for re-election to the same office during the following year.

21.—One [illegible] week before the Annual General Meeting, the Secretaries shall send to each Member a balloting paper containing the names of the Officers and Councillors whom the Council nominate for the ensuing year.

22.—The Society shall elect the Council by ballot, each Member, however, being at liberty to substitute other names for any of those upon the list.

23.—There shall be six Ordinary Meetings annually, of which the first held in each year shall be the Annual General Meeting.

24.—Notices of each meeting, and of the subjects to be considered, shall be sent by the Secretaries to each Member of the Society at least one week before the meeting.

25.—Special Meetings shall be held at the option of the Council, or at the request, in writing, of twenty Members.

26.—The President shall have the power of inviting any person to attend and to take part in the scientific work of the meeting.

27.—If at any time the Council shall be of opinion that the interests of the Society require the expulsion of a Member, they shall submit the question to a special General Meeting, at which, if more than one-half of the Members of the Society vote, by ballot as usual, for the expulsion of the Member, his subscription for the current year shall be returned to him, and he shall thereupon cease to be a Member of the Society.

28.—The Council shall draw up and submit for the approval of the Society, supplementary rules regulating the dates, places, and character of the meetings; shall propose special subjects for investigation by the Society, and shall nominate sub-committees for the methodical carrying out of such investigations. They shall decide, from time to time, on the form of publication which its proceedings are to assume.

29.—No alteration shall be made in the present rules, excepting at the Annual Meeting, or at a Special Meeting convened for the purpose, and unles it be proposed by the Council, or in writing by at least twenty Members, the usual notice be given of the proposed change to every Member before the meeting at which it is to be brought forward

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Each Member receives quarterly, from the beginning of the year in which he is elected, a copy of "BRAIN; a Journal of Neurology," the organ of the Society, and edited for it by Henry Head, M.D., F.R.S.

HONORARY MEMBERS.

Professor WUNDT, Leipzig.
„ HITZIG, Halle.
Sir SAMUEL WILKS, Bart., London.
Dr. J. HUGHLINGS JACKSON, London.

CORRESPONDING MEMBERS.

Professor DEJERINE, Paris.
„ ERB, Heidelberg.
„ GOLGI, Pavia.
„ GRASSET, Montpellier.
„ LUCIANI, Rome.
„ MUNK, Berlin.
„ OBERSTEINER, Vienna.
„ A. PICK, Prague.
„ RETZIUS, GUSTAF, Stockholm.
„ RIBOT, Paris.
Dr. WEIR MITCHELL, Philadelphia.
Dr. ALLEN STARR, New York.

LIST OF PRESIDENTS FROM THE FOUNDATION OF THE SOCIETY.

1886 J. HUGHLINGS JACKSON, M.D., F.R.S.
1887 Sir SAMUEL WILKS, Bart., M.D., F.R.S.
1888 Sir JAMES CRICHTON-BROWNE, M.D., F.R.S.
1889 JONATHAN HUTCHINSON, F.R.S.
1890 THOMAS BUZZARD, M.D.
1891 JOHN S. BRISTOWE, M.D., F.R.S.
1892 H. CHARLTON BASTIAN, M.D., F.R.S.
1893 EDWARD A. SCHÄFER, F.R.S.
1894 DAVID FERRIER, M.D., F.R.S.
1895 Sir WILLIAM H. BROADBENT, Bart., M.D., F.R.S.
1896 ALEXANDER HILL, M.A., M.D.
1897 GEORGE H. SAVAGE, M.D.
1898 Sir VICTOR HORSLEY, M.B., F.R.C.S., F.R.S.
1899 W. H. GASKELL, M.D., F.R.S.
1900 AUGUSTUS WALLER, M.D., F.R.S.
1901 W. JULIUS MICKLE, M.D.
1902 HERBERT WILLIAM PAGE, M.A., F.R.C.S.
1903 J. N. LANGLEY, D.Sc., F.R.S.
1904 S. J. SHARKEY, M.D.
1905 Sir JOHN BATTY TUKE, M.D., LL.D.

LIST OF HUGHLINGS JACKSON LECTURERS.

1897 J. HUGHLINGS JACKSON, M.D., F.R.S.
1900 Geheimrath Professor HITZIG, Halle.
1903 Sir WILLIAM H. BROADBENT, Bart., M.D., F.R.S.

*** Members are requested to communicate with the Secretaries when corrections are necessary.

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EXPLANATION OF ABBREVIATIONS.

O.M., Original Member. | Tr., Treasurer.
Pres., President. | Sec., Secretary.
V.-P., Vice-President. | C., Member of Council.
H.J.L., Hughlings Jackson Lecturer.

ORDINARY MEMBERS.

Elected

1899 Abrahams, Bertram, M.B., F.R.C.P., 14, Welbeck Street, W.
1897 Anderson, H. Kerr, M.D., B.Sc., Whitefield, Great Shelford, Cambs.
1894 Andriezen, W. Lloyd, M.D., 7, Apsley Terrace, Acton, W.
1905 Arkwright, J. A., M.D., 13, Welbeck Street, W.
1903 Armour, D. J., M.B., F.R.C.S., 89, Harley Street, W.
1904 Ascherson, W. Lawrence, M.B., M.R.C.P., St. George's Hospital, W.

1891 Ballance, C. A., M.S., F.R.C.S., 106, Harley Street, W. (C., 1897-1900.)
O.M. Barlow, Sir Thomas, Bart., K.C.V.O., M.D., F.R.C.P., 10, Wimpole Street, W. (Pres., 1906; V.-P., 1904-05.)
1902 Barnes, A. Stanley, M.D., M.R.C.P., 141, Great Charles Street, Birmingham.
O.M. Bastian, H. Charlton, M.D., F.R.C.P., F.R.S., 8a, Manchester Square, W. (Pres., 1892; V.-P., 1891; C., 1886-89, 1893-95.)
1897 Batten, Frederick E., M.D., F.R.C.P., 33, Harley Street, W. (Sec., 1903-06.)
1903 Bayliss, W. M., M.A., D.Sc., F.R.S., St. Cuthbert's, West Heath Road, Hampstead.
O.M. Beach, Fletcher, M.B., F.R.C.P., Winchester House, Kingston Hill, Surrey. (C., 1903-06.)
1903 Beddard, A. P., M.D., F.R.C.P., 44, Seymour Street, Portman Square.
O.M. Beevor, C. E., M.D., F.R.C.P., 135, Harley Street, W. (Tr., 1894-1905; V.P., 1905-06; C., 1893.)
1903 Berry, George A., M.B., F.R.C.S.Edin., 31, Drumsheugh Gardens, Edinburgh.
1898 Bolton, Joseph Shaw, M.D., B.S., B.Sc., County Asylum, Rainhill, near Liverpool.
1896 Bond, C. Hubert, M.D., D.Sc., London County Colony for Epileptics, Ewell, Surrey.
1899 Bond, J. W., M.D., B.S., 26, Harley Street, W.
1892 Bowlby, Anthony A., C.M.G., F.R.C.S., 24, Manchester Square, W.
1898 Boyce, Rubert W., M.B., F.R.S., University College, Liverpool.
1889 Bradford, J. R., M.D., D.Sc., F.R.C.P., F.R.S., 8, Manchester Square, W.

Elected

O.M. BRAMWELL, BYROM, M.D., F.R.C.P.Ed., 23, Drumsheugh Gardens, Edinburgh. (C., 1894, 1903-05.)

1899 BRAMWELL, EDWIN, M.B., C.M., M.R.C.P., 23, Drumsheugh Gardens, Edinburgh.

1895 BRAMWELL, JOHN MILNE, M.B., C.M., 33, Wimpole Street, W.

1892 BRISTOWE, HUBERT CARPENTER, M.D., Wrington, Somerset.

O.M. BROADBENT, Sir WM. H., Bart., K.C.V.O., M.D., F.R.C.P., F.R.S., 84, Brook Street, W. (Pres., 1895; V.-P., 1893; C., 1886-87, 1896.)

1894 BRODIE, T. GRIGOR, M.D., F.R.S., 4, Lancaster Terrace, N.W.

1897 BROWN, H. H., M.D., F.R.C.S., 22, Museum Street, Ipswich.

1888 BROWN, SANGER, M.D., Kenilworth, Chicago, Ill., U.S.A.

O.M. BRUCE, ALEX., M.D., F.R.C.P.Ed., 8, Ainslie Place, Edinburgh. (C., 1895.)

O.M. BRUCE, J. MITCHELL, M.D., F.R.C.P., 23, Harley Street, W.

O.M. BRUNTON, Sir T. LAUDER, M.D., F.R.C.P., F.R.S., 10, Stratford Place, W. (C., 1896-98.)

1895 BRYAN, FREDERICK, M.B., Horton House, Horton, Epsom.

1895 BRYANT, JOHN HENRY, M.D., F.R.C.P., 8, Mansfield Street, Portland Place, W.

1903 BUBB, WILLIAM, M.R.C.S., L.R.C.P., County Asylum, Powick, Worcester.

1890 BURY, JUDSON SYKES, M.D., F.R.C.P., 10, St. John Street, Manchester. (C. 1905-06.)

1900 BUZZARD, E. FARQUHAR, M.D., M.R.C.P., 33, Harley Street, W.

O.M. BUZZARD, THOMAS, M.D., F.R.C.P., 74, Grosvenor Street, W. (Pres., 1890; V.-P., 1888-89; C., 1886-87, 1891-95.)

1898 CALEY, H. A., M.D., F.R.C.P., 24, Upper Berkeley Street, W.

1903 CAMPBELL, A. W., M.D., General Post Office, Sydney, New South Wales.

1898 CAMPBELL, HARRY, M.D., F.R.C.P., 23, Wimpole Street, W.

1899 CHAMBERS, JAMES, M.A., M.D., The Priory, Roehampton.

1891 CLARKE, J. MICHELL, M.D., F.R.C.P., 28, Pembroke Road, Clifton, Bristol. (C., 1903-05.)

1903 CLOUSTON, T. S., M.D., F.R.C.P.Ed., Royal Asylum, Morningside, Edinburgh.

1892 COLE, ROBERT HENRY, M.D., M.R.C.P., 48, Upper Berkeley Street, Portman Square.

1900 COLE, SYDNEY JOHN, M.D. Wilts County Asylum, Devizes.

1899 COLLIER, J. S., M.D., B.Sc., F.R.C.P., 57A, Wimpole Street, W. (C., 1905-06.)

1890 COLMAN, W. S., M.D., F.R.C.P., 9, Wimpole Street, W. (Sec., 1899-1902. C., 1903-05.)

1890 CORNER, HARRY, M.D., Brook House, Southgate, Middlesex.

1899 COUPLAND, SIDNEY, M.D., F.R.C.P., 16, Queen Anne Street, W.

1901 COX, JOSHUA JOHN, M.D., St. Ronan's, Clarendon Road, Eccles, Manchester.

1901 CRAIG, MAURICE, M.D., M.R.C.P., Bethlem Royal Hospital, S.E.

1905 CUNNINGHAM, D. J., M.D., 18, Grosvenor Crescent, Edinburgh.

1898 DARLEY-HARTLEY, W., M.D., M.R.C.P., Main Road, Sea Point, Cape Town, South Africa.

1895 DAVIDSON, ANDREW, M.D., Callan Park, Sydney, N.S.W.

Elected

1897 Dawson, William R., M.D., F.R.C.P.I., Farnham House, Finglas, co. Dublin, Ireland.

1898 Dodd, Henry Work, F.R.C.S., 136, Harley Street, W.

O.M. Dreschfeld, Julius, M.D., F.R.C.P., Stanley House, Wilmslow Road, Withington, Manchester. (V.-P., 1898-99.)

O.M. Drummond, David, M.D., 6, Saville Place, Newcastle-on-Tyne.

1903 Dudgeon, L. S., M.R.C.P., 6, Powis Gardens, London, W.

1892 Dupuy, Eugene, M.D., 53, Avenue Montaigne, Paris.

1899 Fawcett, John, M.D., F.R.C.P., 66, Wimpole Street, W.

O.M. Ferrier, David, M.D., F.R.C.P., F.R.S., 34, Cavendish Square, W. (Pres., 1886; V.-P., 1892-3; C., 1886, 1890-95.)

1903 Flashman, J. F., M.B., Ch.M., B.Sc., Totnes, Temple Street, Petersham, Sydney, Australia.

1903 Fleming, Robert, M.D., F.R.C.P.Ed., 10, Chester Street, Edinburgh.

1903 Fletcher, H. Morley, M.D., F.R.C.P., 98, Harley Street, W.

O.M. Gaskell, W. H., M.D., F.R.S., The Uplands, Great Shelford, Cambs. (Pres., 1899; V.-P., 1897-98; C., 1896, 1900.)

1903 Gibson, G. A., M.D., D.Sc., F.R.C.P.Ed., 3, Drumsheugh Gardens, Edinburgh.

1904 Giglioli Guiolo T., M.D., M.R.C.P., Via Farinata Degli Ulorti, 4, Florence.

O.M. Godlee, R. J., M.B., F.R.C.S., 19, Wimpole Street, W.

1905 Golla, F. Lucien, M.B., B.Ch., National Hospital, Queen Square, W.C.

1899 Goodall, Edwin, M.D., F.R.C.P., Joint Counties Asylum, Carmarthen.

1897 Gossage, Alfred Milne, M.B., F.R.C.P., 54, Upper Berkeley Street, W.

1892 Gotch, Francis, M.A., F.R.S., The Lawn, Banbury Road, Oxford.

1893 Grant, J. Dundas, M.D., F.R.C.S., 18, Cavendish Square, W.

O.M. Gunn, R. Marcus, M.B., F.R.C.S., 54, Queen Anne Street, W. (C., 1899-1900.)

1895 Guthrie, Leonard, M.D., F.R.C.P., 15, Upper Berkeley Street, W. (C., 1904; Sec. 1905-06.)

1902 Hall, W. Hamilton, M.R.C.S., Fordcombe, Tunbridge Wells.

1893 Halliburton, William Dobinson, M.D., F.R.C.P., F.R.S., Church Cottage, 17, Marylebone Road, N.W.

O.M. Hamilton, D. J., M.B., F.R.S.E., University, Aberdeen.

1898 Hanbury, William R., M.R.C.S., L.R.C.P., West Ham Borough Asylum, Goodmayes, Ilford.

1904 Harris, David, F., M.D., The University, St. Andrews.

1897 Harris, Wilfred J., M.D., F.R.C.P., 37, Queen Anne Street, W.

1898 Haslett, John Hanfield, M.R.C.S., L.R.C.P., Halliford House, Sunbury-on-Thames.

1891 Hawkins, Herbert Pennell, M.D., F.R.C.P., 56, Portland Place, W.

O.M. Haycraft, J. B., M.D., D.Sc., F.R.S.E., University College of South Wales, Cardiff.

1891 Head, Henry, M.D., F.R.C.P., F.R.S., 143, Harley Street, W. (C., 1900; Sec., 1901-1904; Editor of "Brain," 1905-06.)

O.M. Hill, Alex., M.A., M.D., Downing Lodge, Cambridge. (Pres., 1896; V.-P., 1895; C., 1892-94.)

Elected

1905 HOBHOUSE, EDMUND, M.D., F.R.C.P., 12, Second Avenue, Brighton (Hove).

1903 HOLMES, GORDON M., M.D., B.Ch., National Hospital, Queen Square, W.C.

O.M. HORSLEY, Sir VICTOR, F.R.C.S., F.R.S., 25, Cavendish Square, W. (Pres., 1898; V.-P., 1896-97; C., 1892-93, 1899.)

1903 HOWLAND, GOLDWIN, W., M.B., M.R.C.P., 540, Spadina Avenue, Toronto, Canada.

1905 HOWELL, C. M. HINDS, M.B., B.Ch., M.R.C.P., 53, Queen Anne Street, W.

1889 HUGGARD, WILLIAM R., M.D., F.R.C.P., Davoz Platz, Switzerland.

1896 HULME, GEORGE FREDERICK, M.B., C.M., South View, Montague Road, Felixstowe.

1904 HUMPHREYS, F. R., M.R.C.S., L.R.C.P., 27, Fellows Road, South Hampstead.

1889 HUMPHRY, LAURENCE, M.D., F.R.C.P., Lensfield, Cambridge.

1903 HUNTER, WALTER K., M.D., D.Sc., 7, Woodside Place, Glasgow.

1901 HUNTER, WILLIAM, M.B., C.M., Government Bacteriological Laboratory, Hong Kong.

1903 HUNTER, WILLIAM, M.D., F.R.C.P., 103, Harley Street, W.

O.M. HUTCHINSON, JONATHAN, F.R.C.S., F.R.S., 15, Cavendish Square, W. (Pres., 1889; V.-P., 1887-88; C., 1886, 1890.)

1889 HYSLOP, T. B., M.D., C.M., M.R.C.P.Ed., Bethlem Royal Hospital, Lambeth, S.E.

O.M. JACKSON, J. HUGHLINGS, M.D., F.R.C.P., F.R.S., 3, Manchester Square, W. (Pres., 1886; H.J.L., 1897; C., 1887, 1892.). Hon. Memb., 1904.

O.M. JESSOP, W. H., M.B., F.R.C.S., 73, Harley Street, W.

1895 JOHNSTON, GEORGE, M.D., M.R.C.P., 3, Montagu Place, Montagu Square, W.

1903 JONES, A. E., M.B., B.Sc., M.R.C.P., 13, Harley Street, W.

1892 JONES, HENRY LEWIS, M.D., F.R.C.P., 143, Harley Street, W.

1894 JONES, ROBERT, M.D., F.R.C.S., Claybury Asylum, Woodford, Essex.

1904 JONES, ROBERT, F.R.C.S.Ed., 11, Nelson Street, Liverpool.

1902 KELYNACK, T. N., M.D., M.R.C.P., 120, Harley Street, W.

1894 KIDD, PERCY, M.D., F.R.C.P., 60, Brook Street, W. (C., 1904-06.)

1896 LANGDON, F. W., M.D., Cincinnati, Ohio, U.S.A.

1898 LANGDON-DOWN, REGINALD L., M.B., M.R.C.P., 47, Welbeck Street, W.

O.M. LANGLEY, J. N., M.A., D.Sc., F.R.S., Hedgerley Lodge, Madingley Road, Cambridge. (Pres., 1903; V.-P., 1901-1902; C., 1898-99, 1904.)

1888 LAWFORD, J. B., M.D., F.R.C.S., 99, Harley Street, W. (C., 1902-3.)

O.M. LEES, D. B., M.D., F.R.C.P., 22, Weymouth Street, W. (C., 1901-1903.)

1897 LEWIS, H. WOLSELEY, M.R.C.S., L.R.C.P., London County Asylum, Banstead, Surrey.

1904 LINDSAY, J. A., M.D., F.R.C.P., 13, College Square E., Belfast.

1897 LOEWENTHAL, MAX, M.D., M.R.C.P., 126, Princes' Road, Liverpool.

1899 LORD, JOHN R., M.B., C.M., London County Asylum, Bexley, Kent.

1898 MCALDOWIE, ALEXANDER M., M.D., 6, Brook Street, Stoke-on-Trent.

Elected

1898 MacCormac, John M'Gee, M.D., 73, Victoria Place, Belfast.

1891 Macdonald, Peter William, M.D., C.M., The County Asylum, Dorchester, Dorset.

1902 McDougall, Wm., M.B., Weald Mount, Haslemere.

1902 Mackenzie, James, M.D., Bank Parade, Burnley.

O.M. Mackenzie, Sir Stephen, M.D., F.R.C.P., 31, Cavendish Square, W. (C., 1896-98.)

1896 Mackintosh, Ashley W., M.A., M.D., 9, Bon Accord Square, Aberdeen.

1903 Macnamara, E. D., M.B., B.C., M.R.C.P., 45, Campden House Road, W.

O.M. Maguire, Robert, M.D., F.R.C.P., 4, Seymour Street, W. (Sec., 1890-93; C., 1893-96.)

O.M. Mann, J. Dixon, M.D., F.R.C.P., 16, St. John Street, Manchester.

1902 Mantle, Alfred, M.D., M.R.C.P., Savile Place, Halifax.

1901 Marris, W. A., M.D., Springfield Road, King's Heath, Birmingham.

1894 Martin, Sidney, M.D., F.R.C.P., F.R.S., 10, Mansfield Street, W.

1905 Mavrogordato, A., M.R.C.S., L.R.C.P., St. Thomas' Hospital, S.E.

1891 May, W. Page, M.D., F.R.C.P., 9, Manchester Square, W. (C., 1906.)

1890 Menzies, William Francis, M.D., B.Sc., M.R.C.P., Cheddleton Asylum, Leek, Staffordshire.

O.M. Mercier, C., M.B., F.R.C.P., Flower House, Southend, Catford, S.E. (C., 1899-1902.)

1897 Meyer, Adolf, M.D., LL.D., Pathological Institute, New York State Hospital, Ward's Island, N.Y., U.S.A.

O.M. Mickle, W. Julius, M.D., F.R.C.P., Grove Hall Asylum, Bow, E. (Pres., 1901; V.-P., 1900; C., 1895-97, 1902-4.)

1895 Moore, Norman, M.D., F.R.C.P., 94, Gloucester Place, W.

O.M. Morris, Malcolm, F.R.C.S.Ed., 8, Harley Street, W.

1902 Morrison, C. S., L.R.C.P.E., County and City Asylum, Hereford.

1888 Mott, F. W., M.D., F.R.C.P., F.R.S., 25, Nottingham Place, W. (Sec., 1894-98; C., 1899-1902.)

1901 Mould, George Wm., M.R.C.S., Oakhurst, Colwyn Bay.

1899 Mould, Gilbert E., M.R.C.S., L.R.C.P., The Grange, nr. Rotherham.

1894 Murray, George R., M.A., M.D., F.R.C.P., 11, Ellison Place, Newcastle-on-Tyne (C., 1906.)

1889 Murray, H. Montague, M.D., F.R.C.P., 25, Manchester Square, W.

1904 Myers, Charles S., M.D., Melrose, Grange Road, Cambridge.

1904 Neild, Newman, M.D., Richmond Hill, Clifton, Bristol.

O.M. Nettleship, Edward, F.R.C.S., Shottermill, Haslemere, Surrey.

1902 Newington, Hayes, F.R.C.P.Ed., The Gables, Ticehurst, Sussex.

O.M. Nicolson, David, C.B., M.D., M.R.C.P.Ed., Balgownie, Guildford, Surrey.

1888 Niermeyer, J. H. H., M.D., Amsterdam.

1904 Nolan, M. J., L.R.C.P., L.R.C.S.I., Down District Asylum, Downpatrick.

1888 Norman, Conolly, F.R.C.P.I., F.R.C.S.I., St. Dymphna's, N. Circular Road, Dublin.

1887 Ogilvie, George, M.B., M.R.C.P., 22, Welbeck Street, W.

1887 Oliver, Thomas, M.D., F.R.C.P., 7, Ellison Place, Newcastle-on-Tyne.

Elected.

O.M. ORANGE, WM., C.B., M.D., F.R.C.P., Oakhurst, Godalming, Surrey.

O.M. ORMEROD, J. A., M.D., F.R.C.P., 25, Upper Wimpole Street, W. (C., 1895-97.)

1899 ORR, DAVID, M.D., County Asylum, Prestwich, Manchester.

1904 OWEN, ARTHUR GEOFFRY, M.D., B.Sc., c/o Prof. Marich, Chanoury University, Melbourne.

1905 OWEN, S. A., M.B., B.C., 50, Harrington Gardens, Kensington, S.W.

O.M. PAGE, HERBERT WILLIAM, M.A., F.R.C.S., 146, Harley Street, W. (Pres., 1902; V.-P., 1900-1; C., 1891-92, 1903-06.)

1903 PALMER, FREDK. S., M.D., M.R.C.P., 19, Harley Street, W.

1895 PARKINSON, JOHN PORTER, M.D., M.R.C.P., 57, Wimpole Street, W.

1901 PARSONS, JOHN HERBERT, D.Sc., F.R.C.S., 27, Wimpole Street, W.

1901 PASSMORE, E. S., M.D., M.R.C.P., Croydon Asylum, Warlingham, Surrey.

1896 PATERSON, DONALD ROSE, M.D., M.R.C.P., 15, St. Andrew's Crescent, Cardiff.

1904 PATON, LESLIE, J., M.B., F.R.C.S., 1, Spanish Place, W.

1905 PIERCE, BEDFORD, M.D., F.R.C.P., The Retreat, York.

1899 PIPER, FRANCIS P., M.B., M.R.C.S., L.R.C.P., King's Leigh, Tankerton, Whitstable.

1888 PITT, G. N., M.D., F.R.C.P., 15, Portland Place, W. (C., 1903-05.)

1899 RAMBAUT, DANIEL F., M.D., B.Ch., County Asylum, Bicton Heath, Shrewsbury.

1894 RANSOM, WILLIAM B., M.D., F.R.C.P., The Pavement, Nottingham.

1890 RAYNER, HENRY, M.D., M.R.C.P.Ed., 16, Queen Anne Street, W.

O.M. REID, E. W., M.B., F.R.S., University College, Dundee.

1897 REISSMANN, CHARLES H., M.B., B.Sc., St. Peter's, Adelaide, South Australia.

1893 RENNIE, GEORGE E., M.D., M.R.C.P., College Street, Sydney.

1895 REYNOLDS, ERNEST S., M.D., F.R.C.P., 2, St. Peter's Square, Manchester.

1891 RIVERS, W. H. R., M.A., M.D., F.R.C.P., St. John's College, Cambridge. (C., 1897-98.)

1905 ROBERTSON-MILNE, CHAS. JOHN, I.M.S., M.B., C.M., Superintendent, Punjab Asylum, Lahore.

1898 ROBERTSON, WILLIAM FORD, M.D., 7, Hill Square, Edinburgh.

1892 ROWE, EDMUND LEWIS, L.R.C.P., L.R.C.S., Borough Asylum, Ipswich.

1901 ROWS, R. G., M.D., County Lunatic Asylum, Lancaster.

1892 RUFFER, MARC ARMAND, M.D., Ramleh, Egypt.

1905 RUSSEL, COLIN K., M.D., C.M., 51, Mackay Street, Montreal, Canada.

1903 RUSSELL, A. E., M.D., M.R.C.P., 9, Wimpole Street, W.

1889 RUSSELL, J. S. RISIEN, M.D., F.R.C.P., 44, Wimpole Street, W. (Sec., 1896-1900; C., 1901-1904.)

1899 RUSSELL, J. W., M.D., M.R.C.P., 72, Newhall Street, Birmingham.

1901 SALAMAN, REDCLIFFE N., M.A., M.B., Frognal End, Frognal Gardens, Hampstead.

O.M. SAVAGE, G. H., M.D., F.R.C.P., 3, Henrietta Street, Cavendish Square. (Pres., 1897; V.-P., 1895; C., 1886-90, 1898.)

1888 SAVILL, T. D., M.D., M.R.C.P., 60, Upper Berkeley Street, W.

Elected

O.M. Schäfer, E. A., F.R.S., University of Edinburgh. (Pres., 1893; V.-P., 1892; C., 1886-98.)

1901 Schorstein, Gustave, M.D., F.R.C.P., 11, Portland Place, W.

O.M. Semon, Sir Felix, C.V.O., M.D., F.R.C.P., 39, Wimpole Street, W. (C., 1899-1901.)

1898 Seward, Wm. Joseph, M.B., Colney Hatch Asylum, N.

O.M. Sharkey, S. J., M.D., F.R.C.P., 22, Harley Street, W. (Pres., 1904; V.-P., 1902-3; C., 1899-92, 1905.)

O.M. Sherrington, C. S., M.D., F.R.S., 16, Grove Park, Liverpool. (V.P., 1906; C., 1892-95.)

1894 Shuttleworth, George E., M.D., Ancaster House, Richmond.

1900 Singer, H. D., M.D., M.R.C.P., Maccagne Buildings, Omaha, Neb., U.S.A.

1895 Smith, F. J., M.D., F.R.C.P., 138, Harley Street, W.

1887 Smith, R. Percy, M.D., F.R.C.P., 36, Queen Anne Street, W. (Editor of "Brain," 1901-1904; C. 1905-06.)

1895 Spicer, W. T. Holmes, M.B., F.R.C.S., 5, Wimpole Street, W.

1901 Stafford, William, M.B., 110, Mansfield Road, Nottingham.

1895 Stanley, Douglas, M.D., M.R.C.P., 21, Broad Street, Birmingham.

1895 Stansfield, Thos. E. K., M.B., London County Asylum, Bexley, Kent.

1892 Starling, Ernest Henry, M.D., F.R.C.P., F.R.S., 40, West End Lane, Hampstead (C., 1904-06.)

1901 Steell, Graham, M.D., F.R.C.P., 96, Mosley Street, Manchester.

1897 Stewart, Purves, M.A., M.D., M.R.C.P., 7, Harley Street, W.

1903 Stewart, T. Grainger, M.B., B.Ch., M.R.C.P., 26, Queen Anne Street, W.

1897 Stirling, Wm., M.D., D.Sc., Owens College, Manchester.

1897 Stoddart, Wm. H., M.D., M.R.C.P., Bethlem Royal Hospital, Lambeth, S.E.

1902 Sullivan, Wm. Charles, M.D., H.M. Prison, Pentonville, N.

1901 Sutherland, Geo. Alexander, M.D., F.R.C.P., 73, Wimpole Street, W.

1888 Syers, Henry W., M.D., M.R.C.P., 75, Wimpole Street, W.

1898 Taylor, F. R. P., M.D., East Sussex Asylum, Hellingly, Sussex.

1891 Taylor, James, M.D., F.R.C.P., 49, Welbeck Street, W. (C., 1900-3 Tr., 1905-06.)

1898 Thomson, H. Campbell, M.D., F.R.C.P., 34, Queen Anne Street, W.

1889 Thorburn, Wm., F.R.C.S., Oxford Lodge, Victoria Park, Manchester. (C., 1901-1903.)

1892 Titchener, E. B., B.A., Ph.D., Cornell Heights, Ithaca, N.Y.

O.M. Tooth, H. H., C.M.G., M.D., F.R.C.P., 34, Harley Street, W. (Sec., 1891-95; C., 1896-99.)

1892 Trevelyan, E. F., M.D., B.Sc., F.R.C.P., 40, Park Square, Leeds.

1892 Tuckey, Charles Lloyd, M.D., 88, Park Street, Grosvenor Square, W.

1892 Tuke, Sir J. Batty, M.D., F.R.C.P.Ed., F.R.S.Ed., 20, Charlotte Square, Edinburgh. (Pres., 1905; V.P., 1903-04.)

1891 Tuke, Thomas Seymour, M.B., Chiswick House, Chiswick.

1891 Turner, William Aldren, M.D., F.R.C.P., 18, Harley Street. (C., 1906.)

Elected

1898 TURNEY, HORACE G., M.D., F.R.C.P., 68, Portland Place, W. (C., 1906.)

O.M. TWEEDY, JOHN, F.R.C.S., 100, Harley Street, W.

1901 VINCENT, SWALE, M.B., Physiological Laboratory, University of Manitoba, Winnipeg, Canada.

1888 VOORTHUS, J. A., M.D., Apeldoorn, 542, Loulaan, Netherlands.

O.M. WALLER, AUGUSTUS, M.D., F.R.S., 32, Grove End Road, N.W. (Pres., 1900; V.-P., 1899; C., 1894-97, 1901.)

O.M. WARNER, FRANCIS, M.D., F.R.C.P., 5, Prince of Wales Terrace, Kensington Palace, W.

1902 WARRINGTON, W. B., M.D., F.R.C.P., 69, Rodney Street, Liverpool.

1904 WATSON, CHALMERS, M.B., F.R.C.P.E., 22, Coates Crescent, Edinburgh.

O.M. WATTEVILLE, A. DE, M.A., M.D., B.Sc., 30, Welbeck Street, W. (C., 1890-95, 1896-1902; Sec., 1886-89; Editor of "Brain," 1880-1900.)

1897 WEBER, F. PARKES, M.D., F.R.C.P., 19, Harley Street, W.

1897 WEST, SAMUEL, M.D., F.R.C.P., 15, Wimpole Street, W.

1897 WHITE, ERNEST W., M.B., M.R.C.P., 40, Margaret Street, Cavendish Square.

O.M. WHITE, W. HALE, M.D., F.R.C.P., 65, Harley St., W. (C., 1897-1902.)

1894 WHITING, ARTHUR J., M.D., M.R.C.P., 142, Harley Street, W.

1889 WIGLESWORTH, JOSEPH, M.D., F.R.C.P., County Asylum, Rainhill, Lancashire.

1905 WILKIN, GRIFFITH CHARLES, M.R.C.S., L.R.C.P.Lond., Conway House, Paignton, Devon.

1900 WILLIAMS, LEONARD, M.D., M.R.C.P., 8, York Street, Portman Square, W.

1894 WILLIAMSON, RICHARD T., M.D., F.R.C.P., 2, St. Peter's Square, Manchester.

1896 WILSON, ALBERT, M.D., 1, Belsize Park, N.W.

1903 WILSON, G. R., M.D., M.R.C.P.Ed., 8, Rutland Square, Edinburgh.

1905 WILSON, S. A. K., M.A., M.B., Ch.B., B.Sc., National Hospital, Queen Square, Bloomsbury.

1896 WOOD, GUY M., M.B., 49, Gordon Square, W.C.

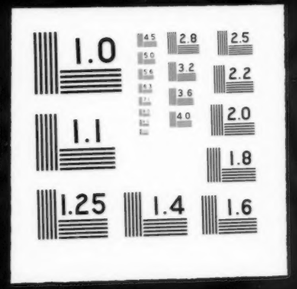
1889 WOOD, T. OUTTERSON, M.D., F.R.C.P.Ed., 40, Margaret Street, Cavendish Square, W.

1892 WOODHEAD, G. SIMS, M.D., F.R.C.P.Ed., 6, Scrope Terrace, Cambridge.

1899 WRIGHT, HAMILTON, M.D., London School of Tropical Medicine.

1898 YOUNG, ROBERT ARTHUR, M.D., B.Sc., F.R.C.P., 41, Wimpole St., W.

RESOLUTION CHART



100 MILLIMETERS

INSTRUCTIONS Resolution is expressed in terms of the lines per millimeter recorded by a particular film under specified conditions. Numerals in chart indicate the number of lines per millimeter in adjacent "T-shaped" groupings.

In microfilming, it is necessary to determine the reduction ratio and multiply the number of lines in the chart by this value to find the number of lines recorded by the film. As an aid in determining the reduction ratio, the line above is 100 millimeters in length. Measuring this line in the film image and dividing the length into 100 gives the reduction ratio. Example: the line is 20 mm. long in the film image, and $100/20 = 5$.

Examine "T-shaped" line groupings in the film with microscope, and note the number adjacent to finest lines recorded sharply and distinctly. Multiply this number by the reduction factor to obtain resolving power in lines per millimeter. Example: 7.9 group of lines is clearly recorded while lines in the 10.0 group are not distinctly separated. Reduction ratio is 5, and $7.9 \times 5 = 39.5$ lines per millimeter recorded satisfactorily. $10.0 \times 5 = 50$ lines per millimeter which are not recorded satisfactorily. Under the particular conditions, maximum resolution is between 39.5 and 50 lines per millimeter.

Resolution, as measured on the film, is a test of the entire photographic system, including lens, exposure, processing, and other factors. These rarely utilize maximum resolution of the film. Vibrations during exposure, lack of critical focus, and exposures yielding very dense negatives are to be avoided.